PREVALENCE OF VITAMIN D DEFICIENCY IN CAD PATIENTS (ACS AND STABLE ANGINA) AND CORRELATION WITH ANGIOGRAPHIC SEVERITY

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CERTIFICATE

This is to certify that the dissertation entitled **– "PREVALENCE OF VITAMIN D DEFICIENCY IN CAD PATIENTS (ACS AND STABLE ANGINA) AND CORRELATION WITH ANGIOGRAPHIC SEVERITY "** is the bonafide original work of **Dr. A. ARVIND** in partial fulfillment of the requirements for D.M.Branch II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held on August 2013. The period of post graduate study and training was from August 2010 to July 2013.

Dr. S. GEETHALAKSHMI, MD
DEAN,
STANLEY MEDICAL
COLLEGE AND HOSPITAL,
CHENNAI - 600001

Prof.Dr.K.KANNAN, MD DM
PROFESSOR AND HEAD OF
DEPARTMENT OF
CARDIOLOGY,
STANLEY MEDICAL
COLLEGE AND HOSPITAL,
CHENNAI - 600001

DECLARATION

I **Dr. A. ARVIND**, solemnly declare that this dissertation entitled **– "PREVALENCE OF VITAMIN D DEFICIENCY IN CAD PATIENTS (ACS AND STABLE ANGINA) AND CORRELATION WITH ANGIOGRAPHIC SEVERITY "** is the bonafide original work done by me at the Department of Cardiology, Stanley Medical College and Government Stanley Hospital during the period 2010-1013 under the guidance and supervision of the Professor and Head of Department of Cardiology of Stanley Medical College and Government Stanley Hospital, **Prof. Dr. K.KANNAN, M.D., D.M.** This dissertation is submitted to THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, towards partial fulfillment of requirement for the award of D.M. Degree (Branch - II) in cardiology.

Place : **Dr. A . ARVIND**

Date :

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TABLE OF CONTENTS

ABBREVIATIONS

AMI	-	Acute Myocardial Infarction
AWMI	-	Anterior Wall Myocardial Infarction
AV	-	Atrioventricular
BMI	-	Body Mass Index
BSA	-	Body Surface Area
CABG	-	Coronary Artery By-Pass Graft
CAD	-	Coronary Artery Disease
CAG	-	Coronary Angiogram
CHF	-	Congestive Heart Failure
CHB	-	complete heart block
CSA	-	chronic stable angina
DVD	-	Double Vessel Disease
EA	-	effort angina
ICCU	-	Intensive Coronary Care Unit
IWMI	-	Inferior Wall Myocardial Infarction
LVEF	-	Left Ventricular Ejection Fraction
MI	-	Myocardial Infarction
MR	-	Mitral Regurgitation
RCA	-	Right Coronary Artery
SD	-	Standard Deviation
SMC	-	Smooth Muscle Cell
SVD	-	Single Vessel Disease
TVD	-	Triple Vessel Disease
VIT d	-	vitamin D
VDBP	-	vitamin D binding protein
VDR	-	vitamin D receptor

INTRODUCTION

Coronary artery disease (CAD),is a leading cause of mortality and morbidity world wide and has reached epidemic proportions. Ischemic heart disease causes 9.4% of total deaths (2.5 million) in less developed countries and 16.3% (1.3 million) of all deaths in well developed countries[1]. The WHO has estimated that in the year 2002 alone, 12.6% of deaths all over the world were due to coronary artery disease[2]. The proportion of these diseases of aging is expected to increase as the world population gets older.

The Indian scenario is similar. Studies have shown that cardiovascular diseases cause about 40% of deaths in the urban centers and 30% of deaths in rural centers in our country[3]. The prevalence of CVD in the adult population has increased in urban centers from around 2% in 1960 to 6.5% in 1970, 7% in the year 1980, to around 9.7% in 1990 and to a significant 10.5% in the year 2000; while in rural centers, it has increased to a lesser extent from about 2% seen in 1970, to 2.5% in 1980, to an estimated 4% in 1990, and finally to a prevalence of 4.5% in 2000[2].

Hence prevention of cardiovascular diseases has become extremely important and elucidation of CVD risk factors and assessing the risk profile of an individual patient is paramount.

Apart from the traditional risk factors, vitamin D has recently emerged as a new risk factor for CVD. It affects multiple homeostatic mechanisms in the body and is involved with metabolic syndrome, hypertension, diabetes mellitus, vascular calcification, inflammation, endothelial dysfunction and atherosclerosis. It is also implicated in the cause of CAD, heart failure, sudden cardiac death and stroke.

In many of the populations studied, the rate of CVD-related death is increased at higher latitudes, is more during the winter. This is believed to be due to the adverse effect of reduced vitamin D levels. Vitamin D levels are known to be lower during the winter season and in high latitudes.

The prevalence of vitamin D deficiency has been estimated to be around 50% in the aged population throughout the world implying a huge global expenditure in health services . It has been estimated that a total of 1 billion of the world's population have reduced levels of the hormone vitamin D .The vitamin D status in Indians is similarly worrying. A study

conducted in Delhi[4] among 1346 healthy subjects of above 50 years showed that vitamin D deficiency was present in 91.2 %.

As vitamin D deficiency is easily correctable, establishing the relationship between vitamin D and risk and severity of atherosclerosis is important.

AIM OF THE STUDY

1. To evaluate the prevalence of vitamin D in the patients attending our hospital- both ACS and chronic stable angina patients.
2. In ACS patients, to correlate the level of vitamin D on admission with clinical endpoints such as the degree of LV dysfunction, killip classification, reinfarction, arrhythmias, the in hospital mortality, and correlate with angiographic lesions such as the number of coronary arteries involved as well as the severity of lesions as measured by the Gensini score.
3. In chronic stable angina patients, to correlate the level of vitamin D with NYHA class & the severity of angiographic lesions as measured by the Gensini score and the number of coronary arteries involved.
4. In all patients to correlate the level of Vit D with the Ankle Brachial Index (ABI) as a measure of peripheral vascular disease.

REVIEW OF LITERATURE

It is estimated that 1 billion people all over the world are suffering from some degree of vitamin D deficiency[5]. We expose only 5% of our skin to the sunlight. Sun exposure is further reduced by age, darker pigmentation, use of sunscreen lotions and increasing latitudes. Thirty minutes of sun exposure over the arms and face, preferably between 10 am to 2 pm daily is adequate to avoid Vitamin D deficiency.

THE INDIAN SCENARIO

India is a tropical country where summer is the predominant season. Despite our climate vitamin D deficiency is rampant in our country. The prevalence of vitamin D deficiency in India has been estimated to be 50-90% according to earlier studies[6].

Harinarayan et al studied the effects of 25(OH)D on BMD and shown that Vit D deficiency is present in 76% of women in reproductive age group and 70% in postmenopausal women[6].

It is a common problem in India despite our sun exposure due to multiple factors such as altered food habits, a high fiber diet which contains phosphates and phytates which lead to depletion of vit D stores and increase our calcium requirement[7], genetic factors such as elevated

25(OH)D-24- hydroxylase[8], and in urban Indians, the number of hours spent indoor has increased plus the increased pollution in urban centers can prevent the ultraviolet rays from adequately synthesizing vit D in the skin[9],repeated pregnancies in already vit D deficient patients aggravate Vit D deficiency both in the mother and the developing fetus.

VITAMIN D PHYSIOLOGY

Vitamin D is a steroid hormone which is obtained through food sources or synthesized in the body. It is an integral part of the "vitamin D-Parathyroid hormone- Calcium" endocrine axis. It is necessary for the optimal absorption of dietary calcium and phosphate and adequate intake of calcium and vitamin D is necessary to maintain the peak bone mass.

The "D" in the vitamin represents either D2 or D3.Vit D2 is called "ergocalciferol" and D3 is called "cholecalciferol". 1,25(OH)2D3 is known as calcitriol. D3 is the most natural form and the type synthesized in humans and found in most healthy fish. D3 is less toxic and more potent form of the vitamin. For fortification purposes vitamin D2 is manufactured by ultraviolet irradiation of ergosterol obtained from yeast, and vitamin D3 is obtained by ultraviolet irradiation of 7-dehydrocholesterol obtained from lanolin.

DIETARY SOURCES OF VITAMIN D

Vitamin D is present in very few foods. Good sources being cod liver oil and sardines, mackerel and salmon.

Food sources of vitamin D

Food types	IU per serving
Cod liver oil ,1 table spoon	1,360
Swordfish, 3 ounces (85 grams)	566
Salmon, 3 ounces (85 grams)	447
Orange juice fortified with vit D, 1 cup	137
Fortified milk, 1 cup	115-124
Liver,3 ounces (85 grams)	42
Egg 1 large	41
Cheese	6

SYNTHESIS OF VITAMIN D

Vitamin D is synthesized primarily from the compound 7-dehydrocholesterol (provitamin D3) which is present in our skin. When skin is exposed to sunlight the 7-dehydrocholesterol in the skin absorbs UV-B rays of wavelength 290-315 nm [10] to form the 9,10-secosterol,known as previtamin D3.This previtamin D3 forms the stable Vitamin D3 by a process known as thermal isomerization.

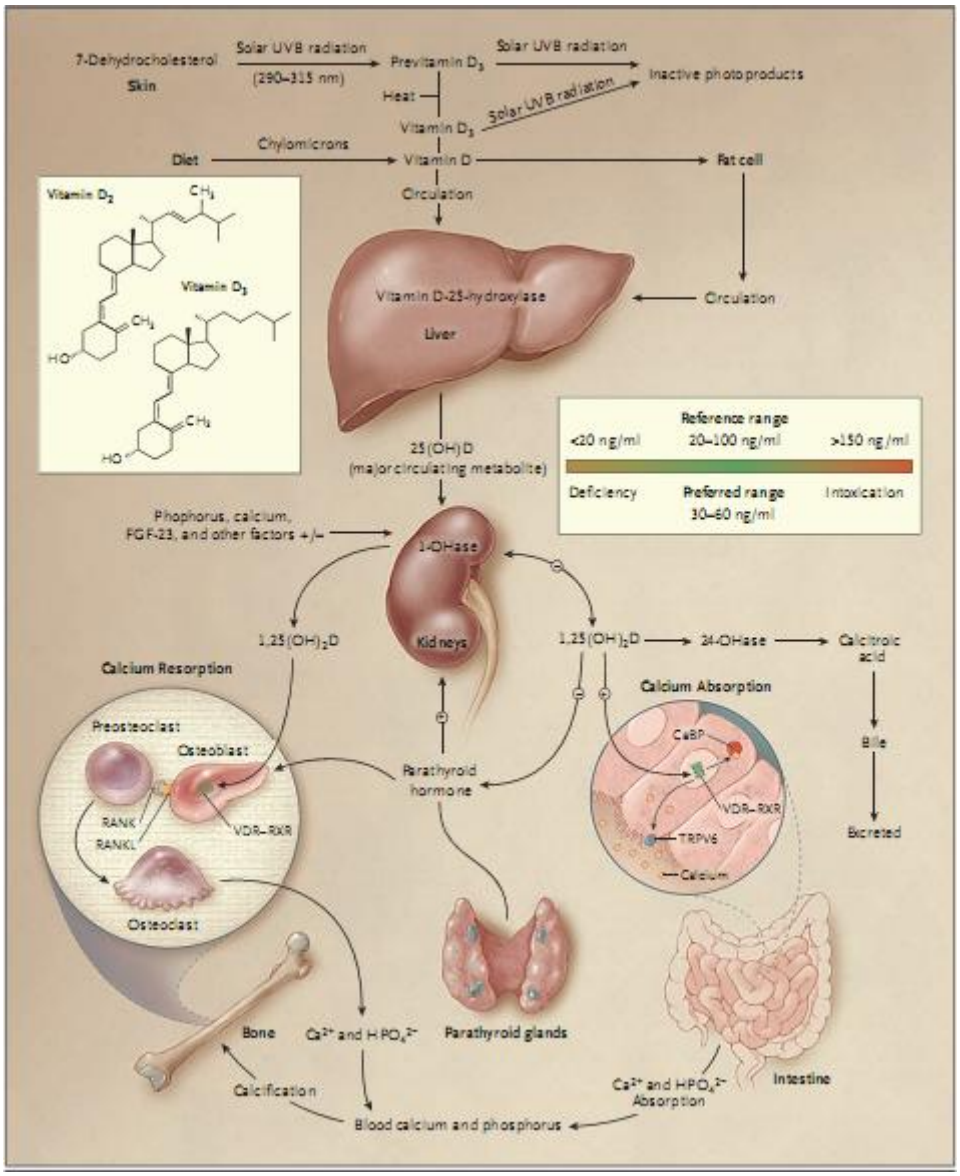
For the metabolic activation of vitamin D to take place, first hydroxylation occurs primarily in the liver. CYP2R1 is the presumed hepatic cytochrome which 25-hydroxylates both forms of vitamin D- D3 and D2.This cytochrome is present predominantly in the liver and testis[11]. It has been shown that 2R1 gene mutations are present in patients with low 25-(OH)D levels and rickets[12].Hence CYP2R1 seems to be the critical in the metabolism of the hormone vitamin D. To assess the vitamin D status ,the commonly used indicator which is measured is plasma 25(OH)D levels[13].

The next step in vitamin D formation is the conversion to[1,25-(OH)2D]. This occurs mainly in the kidney. The renal enzyme 25(OH)D-1-α-hydroxylase mediates the conversion to the activated form of vitamin D- the hormone 1,25(OH)D. 1-α hydroxylase is found in the chromosome 12, the site of defect in (type I vitamin D-dependent rickets or VDDR-I)[14].

But other cells produce vitamin D in conditions such as pregnancy, CKD, sarcoidosis, TB, rheumatoid arthritis and granulomatous disorders. This 1,25 (OH)2D produced outside the kidneys serves a paracrine function. The 1-α hydroxylase enzyme is also found in prostate, breast, colon, monocytes, lungs, pancreas, and in parathyroid cells[15].

Dietary calcium regulates 1-α hydroxylase directly through changes in serum calcium and indirectly by changing parathormone levels[16]. Calcium directly suppresses 1α-hydroxylase activity and mRNA in human cell lines from renal proximal tubules[17]. Parathormone also directly regulates the 1-α hydroxylase enzyme through changes in cAMP[18].

Restricting dietary phosphate increases renal 1-α hydroxylase[19] activity independently of changes in PTH[20] and calcium[21]. The effect of dietary phosphate on 1-α hydroxylase are mediated by phosphaturic factors or phosphatonins, and through fibroblast growth factor 23 and matrix extracellular phosphoglycoprotein (MEPE).



MECHANISMS OF VITAMIN D TRANSPORT

Vitamin D is a fat soluble compound with poor water solubility. Hence it has to be bound to plasma proteins for transport. The most important carrier in the plasma is vitamin D binding protein (DBP). This binds the vitamin D compounds with high affinity in the order of 25(OH)D =24,25(OH)2D > 1,25(OH)2D > vitamin D[22]. >99% of the

circulating vitamin D compounds in the plasma are protein bound, mostly to DBP and to a lesser extent to albumin and lipoproteins. DBP-bound vitamin D metabolites are less prone for metabolism by liver hence they have a longer half life. Only the unbound metabolites can enter the cells to produce their biologic action. Hence DBP prevents vitamin D toxicity.[23]. DBP levels are elevated during pregnancy and estrogen therapy and reduced with liver disease and nephritic syndrome. But the free vitamin D levels do not change when DBP levels alter.

Vitamin D Metabolism

Vitamin D is inactivated inside the cells by vitamin D 24-hydroxylase, which catalyzes oxidation, side chain cleavage followed by inactivation. 24-Hydroxylase activity is increased by phosphate[19,24] and reduced by PTH[25].

MECHANISM OF ACTION

It binds to a high-affinity receptor which functions as a transcription factor. This vitamin D receptor (VDR) belongs to the steroid receptor superfamily. It causes gene transcription by ligand binding followed by heterodimerization with a retinoid X receptor, then binds to vitamin D response elements, and recruits other nuclear proteins. It may

also act additionally through non–VDR pathways or nongenomic VDR-mediated pathways[26,27].

Many tissues possess 1-α-hydroxylase activity. The significance of these paracrine mechanisms of vitamin D production is not clear. This paracrine production may be involved in multiple physiologic functions, such as of cytokines regulation, the renin-angiotensin system, vascular function, immuno modulation, cellular growth as well as differentiation and inflammatory and/or fibrotic pathways[28-34].

RECOMMENDED DAILY INTAKE

Institute of Medicine has recommended the following as adequate daily intake[35]

- for children and in adults up to 50 years -- 200 IU
- for adults between 51 to 70 years -- 400 IU
- for adults above 71 years – 600 IU

But in the absence of enough exposure to sunlight, the requirement for children and in adults rises to approximately 800 to 1000 IU per day[36-41].

One method for vitamin D supplementation to maintain adequate levels is to administer patients one 50,000-IU capsule of D2 given once

a week for total of 8 weeks, then 50,000 IU of vitamin D2 every 2 to 4 weeks[37,42,43].Other strategies being to give 1000 IU of vitamin D3 every day or to give 3000 IU of vitamin D2 daily[37,42,44,45].

Association with disease

The traditional diseases due to vitamin D deficiency such as rickets and osteomalacia are well known. The benefit of vitamin D for prevention of fractures has also been noted[46]. The antifracture effect was dose dependant. Higher doses reduced fractures by 20% in patients >65 years.

The lesser known effects of vitamin D involves the two great killers of the world today such as cardiovascular disease and cancers.

Epidemiologic studies have shown that there is an association between moderate (<15 ng/mL) and severe (<10 ng/mL) levels of vitamin D with increased CV risk[47].

Multiple studies have shown that a 30 -50% higher cardiovascular morbidity and mortality is produced when the exposure to the sun is reduced[48-52].

Vitamin D deficiency is associated with ischemic heart disease , myocardial infarction , hypertension, calcific aortic stenosis and even sudden cardiac death.

Low 25-OH D levels are seen in heart failure[53] and in stroke[54]. vitamin D deficiency has also been found to be associated with metabolic syndrome[55], glucose intolerance[56] and in obesity[56]. Heart failure patients have 34% lower levels of vitamin D when compared to healthy participants[53].

Population based cross sectional study from Scandinavia has shown that serum levels of vitamin D were inversely correlated with blood pressure, triglycerides, and triglyceride removal in an IV fat tolerance test[57]. Serum levels of 25-OH D vitamin D also correlate with fasting insulin, insulin sensitivity, and lipoprotein lipase activity in skeletal muscle and adipose tissue[57]. These findings suggest the role of vitamin D in the metabolic syndrome.

Many epidemiologic studies have shown an association between vitamin D deficiency and cardiovascular mortality. Dobnig et al[58] in 2008 reported a single center study in patients referred for angiogram with a 7.7 year follow up. It showed that lower two quartiles of serum vitamin D had higher all cause and cardiovascular mortality.

Pilz et al[59] in 2008 reported a single center study on 3316 patients referred for angiogram and showed that reduced vitamin D levels were associated with increased strokes both fatal/nonfatal. Wang et al[47] in 2008 reported on a total of 1739 patients with 5.4 years of follow up which showed that low serum vit D levels (<15 ng/ml) was associated with increased CVD events. Melamed et al[60] in 2008 reported on 13,311 patients from the Third National Health and Nutrition Examination Survey with 8.7 year follow up showing that patients with the lowest quartile of serum vit D (<17.8 ng/ml) had higher all-cause mortality than those with the highest quartile.

A prospective nonrandomized study conducted among 1739 patients in the Framingham Offspring Study showed that those with vit D levels (<37.5 nmol/l) had a hazard ratio of 1.62 for myocardial infarction, and heart failure compared with those with vit D levels of at least 37.5 nmol/l[47].The cumulative probability of first cardiac events were substantially higher in hypertensives who were also vitamin D deficient.

In the Health Professionals Follow-up Study , men with low vit D levels (<37.5 nmol/l) had a RR of 2.09 of myocardial infarction when compared with men with sufficient vit D levels[61].

Reduced 25-OH D levels are also related with higher incidences of cancer[62] and immune dysfunction[28]. The LURIC study of 3316 patients that low vitamin D levels independently predicted fatal stroke. Stroke prevention by vitamin D supplementation is an intriguing prospect[63].

VITAMIN D AND THE HEART

The hormone acts on myriad cells involved in cardiovascular homeostasis such as cardiomyocytes, vascular smooth muscle cells and the endothelium. Vitamin D is involved in the development of known cardiac risk factors such as insulin resistance, HT, obesity and metabolic syndrome.

MECHANISMS OF CVD PROTECTION

The exact mechanism by which vitamin D exerts its protective role on the cardiovascular system is yet to be fully elucidated. Vitamin D is a potent hormone which suppresses the renin angiotensin system and in regulation of the blood pressure. It also inhibits thrombosis and arterial calcification. Vitamin D deficiency also increases parathormone levels which have adverse effects on the cardiovascular system.

It reduces inflammation by increasing anti-inflammatory messengers such as IL-10. It can reduce lymphocyte proliferation and

cytokine production. Preclinical and clinical studies have shown a positive effects of vitamin D on endothelial regeneration, smooth muscle cell growth, fibrinolysis, and thrombogenicity[64].

Vitamin D receptors (VDR) are expressed in cardiomyocytes. Studies in rats have shown that vitamin D is protective against myocardial hypertrophy and dysfunction and suppresses RAS and genes involved in cardiac hypertrophy [65-69].

Vitamin D therapy reduces the QTc dispersion, which is a known risk factor for sudden death[70,71].

EXPERIMENTAL DATA IN CARDIOMYOCYTES

Vitamin D affects the growth and proliferation of murine cardiomyocytes. Calcitriol treatment increases the expression of cardiac myotrophin and decreases the expression of ANP - a risk marker with inverse relation to cardiac function. Giving vit D also increases the expression of VDR and causes nuclear localization in these cells[65].

Cardiomyocytes obtained from VDR knockout mice show accelerated contraction and relaxation when compared with wild type mice, and vit D directly affected contractility in the wild type but not in

the knockout cells[66]. Hence we see that 1,25(OH)D is an important hormone which modulates heart cell structure and function.

VDR knockout mice also show hypertrophy of cardiac myofibrils and have underexpression of tissue inhibitors of metalloproteinases[72]. Matrix metalloproteinases (MMPs) are enzymes involved in the myocardial remodelling process. They cause destabilization of atheroma plaques leading to rupture followed by thrombosis. MMPs contribute to progressive ventricular remodelling and dilation, leading to heart failure. In rats who were hypertensive and developed heart failure, 1,25(OH)D treatment caused lower heart weights and collagen levels and less LV diameter when compared with untreated rats[73].

ROLE OF VITAMIN D IN HYPERTENSION

Hypertension is a well known independent risk factor for CVD.

Experimental effects on the vasculature

Vitamin D has a protective role on the vasculature. These beneficial effects include down regulation of MMPs and proinflammatory cytokines such as (IL)-1, IL-6, and (TNF)-α[74,75]. There is also increase in the anti-inflammatory cytokine IL-10 along with vascular calcification inhibitors such as matrix Gla protein (MGP), are also increased along

with type IV collagen present in vascular SMCs. Vitamin D reduces the effect of advanced glycation end products (AGEs) which have a deleterious effect on the endothelial cells.

Mechanism for development of hypertension in vitamin D deficiency is due to the activation of the RAS. Synthesis of renin is increased many times in the renal cells of vitamin D receptor knockout mice[76,77] . VDR-null mice have been shown to have three times higher renin mRNA and the angiotensin II levels which were more than 2.5 times greater than in wild-type mice.

The data on 12 644 patients from the third National Health and Nutrition Examination Survey (NHANES) showed a modest but still significant inverse correlation between serum vitamin D levels and BP after adjustment for other risk factors[78].

ROLE OF VITAMIN D IN LVH

Hypertrophy of the left ventricle (LVH) is a very strong risk factor for cardiovascular mortality. Two clinical trials on hemodialysis patients show that treatment with vitamin D may lead to LVH regression, suggesting a possible cardioprotective action[79,80]. Vitamin D affects the myocardium directly and regulates myocardial hypertrophy, as cardiac hypertrophy is observed in the hearts of VDR knockout mice[81].

Bodyak et al[82] demonstrated that paricalcitol reduced sodium-induced abnormalities of LV in HT rats which were Dahl salt-sensitive.

VITAMIN D AND HEART FAILURE

A small study[75] which included 43 males and 17 females with an ejection fraction of <40% showed that a longer 6-min walk distance correlated with higher 25(OH)D levels. Very low serum 1,25-dihydroxyvitamin D levels (<37.5 pmol/l) have been demonstrated in end-stage heart failure patients[74].

Vitamin D and ECHO abnormalities

Studies have implicated an association between vitamin D deficiency and mitral ring calcification[83]. Calcific aortic stenosis is associated with vitamin D receptor polymorphism[84].

VITAMIN D AND CORONARY ARTERY DISEASE

The vitamin D axis affects inflammation, proliferation of vascular smooth muscle cells and vascular calcification along with blood pressure[85]. All these risk factors affect the development and progression coronary artery disease (CAD). But the data which directly link vitamin D levels to MI are only sparsely available.

A study conducted in Denmark on 128 patients with CAD versus 409 controls shows that vitamin D levels were significantly lower in patients with angina[86]. Another study done in New Zealand[87] on 179 patients with MI showed that MI patients lower mean 25(OH)D levels and that relative risk (RR) of MI decreased across the increasing quartiles of vtamin D (<10 ng/mL the RR was 1 , and in those with 10-13 ng/mL: RR was 0.56 and in those patients with 13.1-16.8 ng/mL: RR was 0.33 and in patients >16.8 ng/mL: RR was 0.30.

One of the mechanisms by which vitamin D affects MI risk is through its effect on calcification. Calcification is commonly seen in atherosclerotic lesions and nearly all of the angiographically significant lesions are calcified[88]. Coronary artery calcification is associated with higher risk of MI[89] and worse 5 year survival[90]. Levels of 1,25-dihydroxyvitamin D are inversely associated with vascular calcification seen in CAD patients[91].

Low vit D is associated with greater inflammation (eg, increased CRP and IL 6 levels and lower IL 10 levels), which could possibly predispose to the increased risk of MI[92-95].

The levels of vitamin D varies among the subgroups in CAD ie, among chronic stable angina, unstable angina and acute MI. In an ACC

moderated poster contribution in ACC 12, a study from Utah[96] analyzed the distribution of vitamin D among the CAD subgroups at presentation. In stable angina patients 38.1% had vitamin D levels of <15 ng/ml and 36.5% had value between16-30 ng/ml and only 37.8% of the patients had a normal value of >30ng/ml. Unstable angina patients had values of <15ng/ml in 35.0%, and 40.7% of patients had a value of 16-30 ng/ml and 44.2% had a normal value of >30 ng/ml. Among MI patients the values were: 26.9% patients had <15ng/ml, 22.7% of patients had a value of 16-30 ng/ml and 18.0% had a normal value of >30 ng/ml. This study concluded that Vit D deficiency is seen to be prevalent among CAD patients regardless of their mode of presentation but severe Vit D deficiency is most prevalent in those with chronic (stable) angina and best predicts risk of future cardiovascular events.

Vitamin D levels and angiographic severity of lesions

Data on the severity of vitamin D deficiency and the extent of angiographic severity of CAD are limited.

In the (LURIC) study , although lower levels of the hormone were associated with heart failure, all-cause and CV mortality, the relationship between vitamin D levels and angiographic severity of CAD was not reported[58,59].

Lee et al[97] reported a higher prevalence of vitamin D deficiency in MI patients who underwent angiograms but the correlation of vitamin D levels with angiographic CAD was not mentioned.

Studies of vitamin D levels in Indian patients with angiographical CAD have reported conflicting results. Even though Shanker et al[98] showed that patients in the lower vitamin D quartile had higher risk for CAD, they did not find an association with severity of CAD.

Rajasree et al[99] reported that the risk of CAD paradoxically increased in patients with vit D levels >89 ng/mL when compared to those with lower levels.

Among Indian patients who undergo coronary angiograms, 80% are vitamin D deficient, while another 13% had insufficiency of vitamin D levels. Only 7% of these patients had normal vitamin D levels[100]. This reflects the high prevalence of vitamin D deficiency in the Indian population in general.

In a study of 3,413 patients with clinically indicated coronary angiography, there was shown to be no significant association between their vitamin D levels and the assessed angiographic severity of CAD[101].

In a Turkish study[102] reported in 2012, low vit D levels were associated with the angiographic severity of lesions as measured by the Gensini score. The Gensini score was negatively associated with serum vitamin D levels ($r = -0.416$, $P < 0.001$).

An Italian study[103] on differential protein expression in MI patients showed that lower levels of vitamin D binding protein in MI patients correlated statistically with the number of involved coronary arteries. The down regulation of vitamin D binding protein is most marked in patients with multivessel disease.

Vitamin D supplementation and cardiovascular risk

The effects of vitamin D supplementation are multiple. Supplementation improves bone strength, and may reduce vascular calcification. Vitamin D has anti-inflammatory properties[104] and supplementation lowered C-reactive protein in 2 small clinical trials[105,106].

The current recommended daily allowance is 200 IU for adults aged from 20-50 years, 400 IU in adults of 51-69 years age, and 600 IU in patients >70 years. But the daily requirement in the older adult which is needed to maximally suppress the parathormone levels is 800 IU per day[107].

Schleithoff et al reported that heart failure patients who were treated with 2000 IU/d of VitaminD had reduced serum TNF levels and increased interleukin-10 (anti-inflammatory cytokine) levels[108].

Witte et al[109] used smaller dose of vitamin D of 400 IU/day but were unable to show a reduction in cytokine levels measured in heart failure. Hence treatment with vitamin D at a dose which is higher than the current recommended daily intake may be needed to reduce the cardiovascular risk.

But those who benefit from supplementation may be those at high risk for developing a CVD event or those with reduced vitamin D levels. Obese individuals have lower levels of vitamin D and improving the hormone levels in these patients may be more beneficial than in patients normal vitamin D levels.

Pfeifer et al[110], have noticed a reduction in BP and the heart rate after the administration of 800 IU of vit D plus 1200 mg of calcium when compared with giving only1200 mg/d calcium to elderly women, suggesting that reduced intake of both vit D and calcium play a contributory role in the development and progression of hypertension and CVD.

Lower mortality has been observed in a vitamin D intervention trial conducted in Britain[111] . In this study the vitamin D group received 2500μg of vitamin D every 4 months over the course of 5 years which is equivalent to 21μg vitamin D per day. Even if statistical significance was not achieved , yet incidence rate ratios were noted to be always close to 1.0, but the mortality rate ratios were always lower, suggesting that the protective effect of vitamin D on cardiovascular and cancer mortality is more effective than its effect on disease incidence.

MATERIALS AND METHODS

PATIENTS

This study was conducted from March 2012 to February 2013. The study population includes patients admitted to our ICCU with Acute Coronary Syndrome (ACS) and patients with effort angina (EA)who underwent coronary angiogram. The diagnoses were confirmed by experienced cardiologists and angiograms were performed as indicated by the patient's clinical profile and presentation according to established guidelines.

Inclusion criteria

- Age from 18 – 75 years
- Acute coronary syndrome patients being admitted to our ICCU
- Chronic stable angina patients undergoing angiogram

Exclusion criteria

- chronic kidney disease patients
- patients with known diseases involving the Parathormone – vit D axis such as hyper/hypo parathyroidism, hyper/ hypovitaminosis D

- patients who are on calcium and vitamin D /parathormone supplementation.

Data collection technique and tools

Ethical issues:

As this study involves the taking of blood investigations, and an invasive diagnostic procedure such as coronary angiogram, all patients and their relatives were explained the study design at the time of enrollment.

Informed consent was obtained from all patients at the time of admission (a copy is enclosed).

The following demographic and clinical data were obtained as soon as the patient was admitted and enrolled.

A proforma (a copy enclosed) for each patient was filled which included

Detailed clinical history :

- Age of the patient
- Symptom history

- A complete risk factor profile (including diabetes, hypertension, dyslipidemia, smoking, family history, prior history of CAD) .
- physical examination
    - Including the pulse rate, blood pressure, cardiac & respiratory system clinical examination,
    - Anthropometric measurements such as the height, weight,body mass index,

Investigations

- A 12 lead electrocardiogram was obtained. ECG using right sided leads or posterior leads was taken if necessary.
- Echocardiogram was performed at time of admission and prior to discharge.
- Complete blood investigations including blood counts, blood sugar, urea and creatinine , liver function test, HBsAg, HCV, and HIV were taken at admission.
- Blood levels of vitamin D were assessed from samples taken at time of admission
- e GFR was calculated by the Cockroft Gault formula
- ABI was calculated at time of admission

- Chest x ray PA view
- Coronary angiogram performed during index hospitalization or as a delayed strategy as may be warranted.

Echocardiography was performed using Aloka model no: IPC - 1530 by experienced cardiologists.

SAMPLE COLLECTION

Blood samples were obtained on admission. 2 ml of non heparinised blood was collected under strict aseptic precautions from the antecubital vein and refrigerated. This sample was sent to the laboratory where it was centrifuged to separate the serum. The method of assay is the Chemiluminescence immunoassay (CLIA) using a fully automated Diasorin system.

Vitamin D deficiency (<30ng/ml) is further graded according to serum 25-OH D levels as deficient, insufficient, hypovitaminosis.

Range of Vitamin D levels[112-114]

25-OH D Level	ng/mL
Deficient	<10
Insufficient	10-20
Hypovitaminosis	20-30
Adequate	30-100
Toxic	>100/140


LIAISON

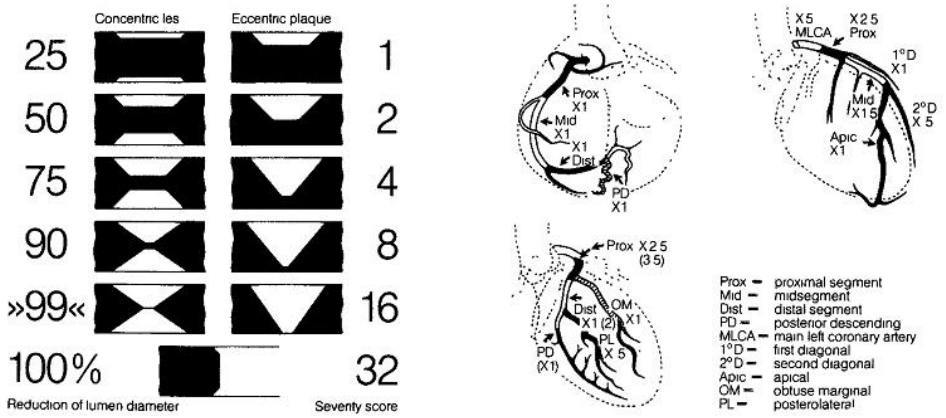
A vitamin D level of more than 30 ng /ml indicates adequate levels of vitamin D[115]. The vitamin D status is best reflected by serum 25-OH D levels since this compound has a half life lasting several weeks. The biologically active form which is 1,25-(OH)2- vitamin D is not routinely measured as this has a short half life lasting for only a few hours. The vitamin D levels are expressed in two units, either in International Units (IU) or nanograms(ng) /ml. One microgram is equivalent to 40 IU.

Coronary angiogram was done at a median of 1 day (range 0 to 5 days) after admission using the Siemens 2000. The studies were performed and reviewed by a team of cardiologists with advanced training in the catheterization laboratory.

The Gensini score[116] was used to calculate the severity of angiographic lesions and verified by a team of experts. This score assigns heavier weight to more severe luminal narrowings and also gives weight to the segment of coronary artery involved according to vessel size and importance; segments that serve larger regions are more heavily weighted.

A 25% diameter reduction is given a score of 1. Likewise diameter reduction is scored as following; 50% reduction as 2; a 75% reduction as

4; a 90% reduction as 8;a 99% reduction is scored as 16 and 100% cut off is scored as 32. This score is multiplied by the weight given to coronary segments such as Left main 5; prox LAD is given 2.5; mid LAD 1.5; distal LAD 1;the first diagonal is given 1 and second diagonal is given 0.5; prox LCX is given 2.5 if nondominant and 3.5 if dominant; distal LCX is given 1 (nondominant) and 2 (dominant). OM s 1;PLB 0.5;PDA 1;RCA 1each for prox ,mid,and distal. The diameter reduction score is multiplied by the coronary segment score for each lesion. The total gives the Gensini score for the patient.



Analysis of the Results

The data collected during the study period was compiled, analyzed and interpreted according to the objectives and parameters that had been decided.


H: 55 %
F: 30 %

Statistical Analysis

All statistical analysis were done with SPSS version 16.0 for windows. The data are expressed as Mean ± SD for quantitative data. The qualitative data are expressed as frequency and percentage. The differences between multiple groups were analyzed using the student t test and Annova. The descriptive data were used for chi-square analysis. The probability value less than 0.05 was considered significant.

Pearson chi square test was used to compare vitamin D levels with all the parameters including the angiographic distribution of lesions and severity as per the Gensini score.

RESULTS

Data Analysis

Table 1: vitamin D x Sex

Vitamin D ng/ml	Male	Female	Total	P Value
>30	12	3	15	Chi-square=1.08 Df=3 p=0.80 (Not significant)
20-30	27	7	34	
10-20	35	5	40	
<10	16	3	19	
TOTAL	90	18	108	
Mean	18.93±8.39	20.50±8.66	19.00±8.42	t=0.83 df=106 p=0.41 Not Significant

Chi-square=1.08 with 3 degrees of freedom. The association between vitamin D levels and the sex of the patient was not significant (p=0.80).

Table 2: **vitamin D x Age**

Base line Characteristics:

	Age (Mean ± SD)
Male	52.09 ± 10.29
Female	54.22 ± 9.42
Total	52.41 ± 10.14

t = 0.81 df=106 p=0.42 Not significant

vitamin D ng/ml	<40yrs	40-60yr	>60yrs	Total	P Value
>30	2	10	3	15	Chi-square 2.14 Df= 6 p=0.91 Not Significant
20-30	7	20	7	34	
10-20	4	26	10	40	
<10	2	13	4	19	
Total	15	69	24	108	
Mean	20.79±7.98	18.68±8.70	18.81±8.06	19.04±8.42	F=0.39 df=2,105 p=0.68 Not Significant

Chi-square=2.14 with 6 degrees of freedom. The association between vitamin D levels and the age of the patient was not significant (p=0.91)

Table 3: vitamin D x Diagnosis

vitamin D ng/ml	ACS	EA	Other indications	Total	P Value
>30	10	3	2	15	Chi square=13.37 Df=6 P=0.04 Significant
20-30	18	15	1	34	
10-20	27	13	0	40	
<10	16	3	0	19	
Total	71	34	3	108	

Chi-square=13.37 with 6 degrees of freedom. The association between vitamin D levels and the presentation of the patient was **significant** (p=0.04)

Table 4: vitamin D x Smoking

vitamin D ng/ml	Smoker	Non smoker	Total	P Value
>30	5	10	15	Chi square=1.55 Df=3 P=0.67 Not Significant
20-30	13	21	34	
10-20	17	23	40	
<10	10	9	19	
Total	45	63	108	
Mean	19.61±8.46	18.11±8.38		t=0.93 df=106 p=0.36 Not Significant

Chi-square=1.55 with 3 degrees of freedom. The association between vitamin D levels and smoking was not significant (p=0.67).

Table 5: vitamin D x SHT

vitamin D ng/ml	Hypertensive	Not known Hypertensive	Total	P Value
>30	3	12	15	Chi square=4.23 Df=3 P=0.24 Not Significant
20-30	14	20	34	
10-20	20	20	40	
<10	9	10	19	
Total	46	62	108	
Mean	17.32±7.89	20.25±8.65		t=1.81 df=106 p=0.07 Not Significant

Chi-square=4.23 with 3 degrees of freedom. The association between vitamin D levels and HT was not significant (p=0.24).

Table 6: vitamin D x Diabetes

vitamin D ng/ml	Diabetics	Non Diabetics	Total	P Value
>30	5	10	15	Chisqure=3.14 Df=3 P=0.37 Not significant
20-30	11	21	34	
10-20	19	23	40	
<10	10	9	19	
Total	63	45	108	
Mean	19.78±8.56	17.64±8.13		t=1.43 df=106 p=0.16 Not Significant

Chi-square=3.14 with 3 degrees of freedom. The association between vitamin D levels and DM was not significant (p=0.37)

Table 7: vitamin D x Dyslipidemia

vitamin D ng/ml	Present	Absent	Total	P Value
>30	6	9	15	Chi square=5.58 Df=3 P=0.13 Not Significant
20-30	12	22	34	
10-20	24	16	40	
<10	11	8	19	
Total	53	55	108	
Mean	17.67±7.77	20.29±8.88		t=1.63 df=106 p=0.11 Not Significant

Chi-square=5.58 with 3 degrees of freedom. The association between vitamin D levels and dyslipidemia was not significant (p=0.13)

Table 8: vitamin D x Obesity

vitamin D ng/ml	Present	Absent	Total	P Value
>30	2	13	15	Chi square =7.33 Df=3 P=0.06 Not Significant
20-30	0	34	34	
10-20	8	32	40	
<10	3	16	19	
Total	13	95	108	
Mean	15.75±9.11	19.45±8.27		t=1.49 df=106 p=0.14 Not Significant

Chi-square=7.33 with 3 degrees of freedom. The association between vitamin D levels and obesity was not significant (p=0.06)

Table 9: vitamin D x NYHA Classification

vitamin D ng/ml	0	II	III	IV	Total	P Value
>30	12	3	0	-	15	Chi square=7.61 Df=6 P=0.27 Not significant
20-30	21	9	4	-	34	
10-20	25	6	9	-	40	
<10	15	2	2	-	19	
Total	73	20	15		108	

Chi-square=7.61 with 6 degrees of freedom. The association between vitamin D levels and the NYHA functional class was not significant (p=0.27)

Table 10: vitamin D x Killip Classification

vitamin D ng/ml	1	2	3	4	Total	P Value
>30	6	0	0	1	7	Chi square=10.21 Df=9 P=0.33 Not Significant
20-30	11	3	1	0	15	
10-20	12	3	4	0	19	
<10	8	1	3	2	14	
Total	37	7	8	3	55	

Chi-square=10.21 with 9 degrees of freedom. The association between vitamin D levels and Killip Classification was not significant (p=0.33).

Table 11: vitamin D x Inotropic Support

vitamin D ng/ml	YES	NO	Total	P Value
>30	1	14	15	Chisquare=9.54 Df=3 P=0.02 significant
20-30	1	23	34	
10-20	6	34	40	
<10	6	13	19	
Total	14	94	108	

Chi-square=9.54 with 3 degrees of freedom. The association between vitamin D levels and Inotropic Support was **significant** (p=0.02)

Table 12: vitamin D x Death

vitamin D ng/ml	YES	NO	Total	P Value
>30	1	14	15	Chisquare=11.00 Df=3 P=0.01 significant
20-30	0	34	34	
10-20	1	40	40	
<10	3	16	19	
Total	4	104	108	

Chi-square=11 with 3 degrees of freedom. The association between vitamin D levels and death was **significant** (p=0.01).

Table 13: vitamin D x Re MI

vitamin D ng/ml	YES	NO	Total	P Value
>30	0	15	15	Chisquare=10.73 Df=3 P=0.01 significant
20-30	1	33	34	
10-20	1	39	40	
<10	4	15	19	
Total	6	102	108	

Chi-square=10.73 with 3 degrees of freedom. The association between vitamin D levels Re infarction was **significant** (p=0.01).

Table 14: vitamin D x Arrhythmia

vitamin D ng/ml	YES	NO	Total	P Value
>30	1	14	15	Chi square=22.32 Df=3 P=0.000 significant
20-30	3	31	34	
10-20	11	29	40	
<10	12	07	19	
Total	27	81	108	

Chi-square=22.32 with 3 degrees of freedom. The association between vitamin D levels and arrythmia was **significant** (p=0.000).

Table 15: vitamin D x Left Ventricular Dysfunction

vitamin D ng/ml	YES	NO	Total	P Value
>30	8	7	15	Chi square=4.54 Df=3 P=0.21 Not significant
20-30	20	14	34	
10-20	26	14	40	
<10	16	3	19	
Total	70	38	108	

Chi-square=4.54 with 3 degrees of freedom. The association between vitamin D levels and LV dysfunction was not significant (p=0.21)

Table 16: **vitamin D x MR**

vitamin D ng/ml	Mild	moderate	trivial	none	Total	P Value
>30	3	1		11	15	Chi square=10.07 Df=6 P=0.12 Not significant
20-30	13	1		20	34	
10-20	22	3		15	40	
<10	12	1		6	19	
Total	50	6		52	108	

Chi-square=10.07 with 6 degrees of freedom. The association between vitamin D levels and Mitral regurgitation was not significant (p=0.12).

Table 17: vitamin D x aortic sclerosis

vitamin D ng/ml	YES	NO	Total	P Value
>30	4	11	15	Chi saqure=4.81 Df=3 P=0.18 Not significant
20-30	9	25	34	
10-20	17	23	40	
<10	10	09	19	
Total	40	68	108	

Chi-square=4.81 with 3 degrees of freedom. The association between vitamin D levels and aortic sclerosis was not significant (p=0.18).

Table 18: vitamin D x Diastolic Dysfunction

vitamin D ng/ml	Normal	I	II	III	Total	P Value
>30	6	8	0	1	15	Chi square=30.31 Df=9 P=0.000 significant
20-30	5	25	2	2	34	
10-20	2	29	2	7	40	
<10	0	11	6	2	19	
Total	13	73	10	12	108	

Chi-square=30.31with 9 degrees of freedom. The association between vitamin D levels diastolic dysfunction was significant (p=0.000).

Table 19: vitamin D x normal coronaries

vitamin D ng/ml	YES	NO	Total	P Value
>30	3	12	15	Chi square=9.40 Df=3 P=0.02 significant
20-30	8	26	34	
10-20	1	39	40	
<10	1	18	19	
Total	13	95	108	

Chi-square=9.4 with 3 degrees of freedom. The association between vitamin D levels normal coronaries in angiogram was **significant** (p=0.02).

Table 20: vitamin D x SVD

vitamin D ng/ml	YES	NO	Total	P Value
>30	8	7	15	Chi square=6.99 Df=3 P=0.07 Not significant
20-30	13	21	34	
10-20	10	30	40	
<10	3	16	19	
Total	34	74	108	

Chi-square=6.99 with 3 degrees of freedom. The association between vitamin D levels single vessel disease on angiogram was not significant (p=0.07).

Table 21: vitamin D x DVD

vitamin D ng/ml	YES	NO	Total	P Value
>30	2	13	15	Chi square=4.77 Df=3 P=0.19 Not significant
20-30	10	24	34	
10-20	13	27	40	
<10	2	17	19	
Total	27	81	108	

Chi-square=4.77 with 3 degrees of freedom. The association between vitamin D levels and double vessel disease on angiogram was not significant (p=0.19).

Table 22: vitamin D x TVD

vitamin D ng/ml	YES	NO	Total	P Value
>30	2	13	15	Chi square=23.74 Df=3 P=0.000 significant
20-30	3	31	34	
10-20	18	24	40	
<10	13	6	19	
Total	34	74	108	

Chi-square=23.74 with 3 degrees of freedom. The association between vitamin D levels and triple vessel disease on angiogram was **significant** (p=0.000).

Table 23: vitamin D x Lt main

vitamin D ng/ml	YES	NO	Total	P Value
>30	0	15	15	Chi square=12.31 Df=3 P=0.01 significant
20-30	1	33	34	
10-20	5	35	40	
<10	6	13	19	
Total	12	96	108	

Chi-square=12.31 with 3 degrees of freedom. The association between vitamin D levels left main involvement in angiogram was **significant** (p=0.01).

Table 24: vitamin D x 100% cut off

vitamin D ng/ml	YES	NO	Total	P Value
>30	1	14	15	Chi square=18.19 Df=3 P=0.000 significant
20-30	2	32	34	
10-20	10	30	40	
<10	10	9	19	
Total	23	85	108	

Chi-square=18.19 with 3 degrees of freedom. The association between vitamin D levels 100% cut off in angiogram was **significant** (p=0.000).

Table 25: vitamin D x gensini score

vitamin D ng/ml	Gensini score	P Value
>30	21.25 ± 20.06	F=8.64 Df=(3,91) P=0.000 significant
20-30	25.42 ± 23.97	
10-20	42.50 ± 25.42	
<10	62.00 ± 35.83	
Total	38.84 ± 29.85	

The association between vitamin D levels and Gensini score was **significant** (p=0.000).

Table 26: vitamin D x eGFR

vitamin D ng/ml	eGFR>60ml/min	eGFR<60ml/min	Total	P Value
>30	15	0	15	Chi square=3.46 Df=3 P=0.33 Not significant
20-30	34	0	34	
10-20	38	2	40	
<10	19	0	19	
Total	106	2	108	

Chi-square=3.46 with 3 degrees of freedom. The association between vitamin D levels and eGFR was not significant (p=0.33).

Table 27: vitamin D x ABI

vitamin D ng/ml	ABI>1	ABI 0.9-1	ABI<1	Total	P Value
>30	8	7	0	15	Chi square=6.51 Df=6 P=0.37 Not Significant
20-30	8	24	2	34	
10-20	10	28	2	40	
<10	5	12	2	19	
Total	31	71	6	108	

Chi-square=6.51 with 6 degrees of freedom. The association between vitamin D levels and ABI was not significant (p=0.37).

DISCUSSION

This study was conducted to assess if vitamin D levels correlate with the atherosclerotic burden, number of coronary arteries involved and the angiographic severity of lesions as calculated by the Gensini score in both ACS and chronic stable angina patients. The association between vitamin D levels and the in hospital course and complications of Acute Coronary Syndrome patients was assessed. In all patients the relation between vitamin D levels and clinical and echocardiographic parameters were assessed.

Vitamin D levels on comparison with clinical characteristics

Gender vs vitamin D levels

- In our study population of 108 patients, 83% were males 17% were females. This reflects the increased prevalence of CAD in males
- The p value was 0.8 and no association was seen between sex of the patient and vitamin D levels.
- 13% of males and 16 % of females had an adequate vitamin D level >30ng/ml.

Age Group Vs vitamin D levels

- The mean age of the males in our study was 52.09 ± 10.29 and the females was 54.22 ± 9.42.
- The patients were divided into three groups of <40 years,40-60 years and >60 years .The majority of our patients were >40 years (86%) reflecting the age related risk of atherosclerosis. The mean vitamin D levels did not vary significantly among the three groups but was consistently insufficient in all the age groups. The mean vitamin D levels for the three groups were 20.79±7.98, 18.68±8.70, 18.81±8.06 respectively.
- The p value was 0.91 and there was no association between vitamin D levels and age groups . Vitamin D was deficient across all age groups.

vitamin D levels in the entire study population

- The mean vitamin D level in the entire study population was insufficient (19.00±8.42).
- Only 13% of the study population irrespective of the age and sex had an adequate vitamin D level of >30ng/ml.

- Rest of the study population (87%) were vitamin D deficient to varying degrees
    - 17.5% were severely deficient (<10ng/ml). 31% had hypovitaminosis (20-30ng/ml) and 37% had insufficient vitamin D levels (10-20ng/ml)
- This reflects the high degree to which the patient population undergoing coronary angiogram is deficient in vitamin D.

vitamin D levels in ACS and CSA patients

- 65% of our study group were Acute Coronary Syndrome (ACS) patients
- 31% were chronic stable angina patients (CSA)
- The association between the vitamin D levels and the presentation of the patient was **significant**(P=0.04)
- On analyzing the vitamin D level subgroups in those with ACS patients, only 14% had adequate vitamin D levels (>30ng/ml), another 25 % had hypovitaminosis D (20-30ng/ml), 38 % had insufficient vitamin D levels (10-20ng/ml) and another 22 % had <10 ng/ml.
- Of the total no of patients in our study who had vitamin D level of <10ng/ml , a significant 84% had an ACS

- On analyzing the vitamin D level subgroups in those with EA patients, only 8 % had adequate vitamin D levels (>30ng/ml), another 44 % had hypovitaminosis D (20-30ng/ml), 38 % had insufficient vitamin D levels (10-20ng/ml) and another 8 % had <10 ng/ml.
- Hence we see that 86% of ACS patients had inadequate vitamin D levels and that a significant 22% of ACS were in the lowest quartile of vitamin D level(<10ng/ml) compared to EA/chronic stable angina patients who had inadequate vitamin D levels in 92% and that only 8% were in the lowest quartile of vitamin D level(<10ng/ml)

Vitamin D levels vs smoking

- 41% of our study population were smokers. The mean vitamin D levels in smokers and non-smokers were 19.61±8.46 and 18.11±8.38 respectively.
- Inadequate vitamin D levels were seen irrespective of the smoking status and there was no statistical association between smoking status and vitamin D levels (P=0.67)
- Of those with adequate vitamin D levels (>30ng/ml) 66% were non-smokers and 33.3% were smokers.Only 11% of

smokers had adequate vitamin D levels compared to 15% of non-smokers.

Vitamin D levels vs hypertension

- 42% of our study population were hypertensives. The mean vitamin D level in them was 17.32±7.89 compared to 20.25±8.65 in nonhypertensives. Both had reduced vitamin D levels in the insufficient range and the difference between their levels was not statistically significant (P=0.24) probably because of the high prevalence of vitamin D deficiency in the population undergoing angiography[100].
- Of those with adequate vitamin D levels (>30ng/ml) 80% were non hypertensives and only 20% were hypertensives.
- Only 6% of hypertensives had adequate vitamin D levels compared with 19.3% of non hypertensives.
- the hypertensive population was less likely to have a normal range of vitamin D level (>30ng/ml).This is consistent with the effect of vitamin D on the vasculature[76-78].

Vitamin D levels vs Diabetes Mellitus

- of our total study population, were diabetics. The mean vitamin D levels in diabetics and nondiabetics were and respectively. There was no statistical significance in the vitamin D levels between the two populations (P=0.37).
- both diabetics and nondiabetics had reduced vitamin D levels in the insufficient range.
- Of those with adequate vitamin D levels (>30ng/ml) were nondiabetics and were diabetics.

Vitamin D levels vs dyslipidemia

- of our total study population, 49% had dyslipidemia. The mean vitamin D levels in those with dyslipidemia and without dyslipidemia were 17.67±7.77 and 20.29±8.88 respectively. There was no statistical significance in the vitamin D levels between the two populations(P=0.13).
- both with dyslipidemia and without dyslipidemia had reduced vitamin D levels in the insufficient range.
- 11% of those with dyslipidemia had adequate vitamin D levels (>30ng/ml) while 16% of those without dyslipidemia had adequate vitamin D levels. Of those with adequate

vitamin D levels (>30ng/ml) 60% were without dyslipidemia and 40% had dyslipidemia.

- 20% of those with dyslipidemia had vitamin D levels below 10ng/ml compared to 14% in without dyslipidemia.
- Hence even though both groups had low vitamin D levels , it is seen that those with dyslipidemia were less likely to have adequate vitamin D levels and more likely to have vitamin D <10 ng/ml

vitamin D levels vs obesity

- of our total study population, 12% had obesity. The mean vitamin D levels in obese and non obese were 15.75±9.11 and 19.45±8.27 respectively. There was no statistical significance in the vitamin D levels between the two populations(P=0.06).

vitamin D levels vs NYHA Classification

- 57% of effort angina patients were in NYHA class II and 43 % were in NYHA class III. There was no statistical significance in the vitamin D levels between the two functional classes(P=0.27).

- None of the patients with functional class III had adequate vitamin D levels compared to 15% of those with functional class II.

vitamin D levels vs Killip Classification

- 67% of ACS patients were in Killip I. There was no statistical significance between the vitamin D levels and the Killip class (P=0.33)
- But none of the patients in Killip II & III had adequate vitamin D(>30ng/ml) levels compared to 16% in those presenting with Killip I. 66% of Killip IV patients had vitamin D levels <10 ng/ml.

vitamin D levels vs Ionotropic support

- The association between the vitamin D levels and ionotropic support was **significant**(P=0.02)
- of our total study population, 12% required ionotropic support
- Of those with adequate vitamin D levels (>30ng/ml) 93% did not require any ionotropic support.

- On analyzing the vitamin D level subgroups in those requiring ionotropic support, only 7% had adequate vitamin D levels (>30ng/ml), another 7% had hypovitaminosis D (20-30ng/ml), 42% had insufficient vitamin D levels (10-20ng/ml) and another 42% had <10 ng/ml.
- Of the total no of patients with <10ng/ml , 31% required ionotropic support compared to 15% in those with 10-20 ng/ml and only 2% of those with 20-30 ng /ml required ionotropic support.
- Hence we see that those with lower vitamin D levels were more likely to require ionotropic support.This is probably due to the greater severity of atherosclerosis lesions and larger infarcts.

vitamin D levels vs Death

- 3% of our total study population died.
- The association between the vitamin D levels and mortality was **significant**(P=0.01)
- On analyzing the vitamin D level subgroups in those who died, 75% had a vitamin D level of less than 20 ng/ml. Of

those with vitamin D levels between 10-20ng/ml, 2 % died while 15% of those with< 10 ng/ml died.

- Hence we see that mortality was directly related to lower vitamin D levels and significantly higher in those with the lowest levels (<10ng/ml)

vitamin D levels vs Re MI

- 5% of our study population experienced re infarction
- The association between the vitamin D levels and re infarction was **significant**(P=0.01)
- None of those who had re infarction had adequate vitamin D levels (>30ng/ml).66% of those who developed re infarction had vitamin D levels of <10ng/ml. Of those with vitamin D levels of <10ng/ml , 21% developed reinfarction, while only 2% of those with levels between 10-20ng/ml and 2% in those with 20-30ng/ml developed re infarction.
- Hence we see that reinfarction is directly related to lower vitamin D levels and those with the lowest vitamin D levels had the most reinfarction.

vitamin D levels vs arrythmias

- 25% of our study population developed arrhythmias.
- The association between the vitamin D levels and arrythmias was **significant**(P=0.000)
- Of those with adequate vitamin D levels (>30ng/ml), only 6% developed arrhythmias, while in those with <10 ng/ml , 63% developed arrhythmias.
- Of the total no of patients who developed arrhythmias, 3% had a vitamin D level of >30 ng/ml, 11% had vitamin D level of 20-30 ng/ml and 40% had a vitamin D level of 10-20 ng/ml and 44 % had a vitamin D level of <10 ng/ml
- While only 6% of those with vitamin D level of >30ng/ml developed arrhythmias,8% of those with vitamin D level of 20-30 ng/ml and 27% of those with vitamin D level of 10-20 ng/ml and 63% of those with vitamin D level of <10 ng/ml developed arrhythmias.
- From this data we see that the incidence of arrhythmias rises with falling vitamin D levels and is highest in those with lowest vitamin D levels.

vitamin D levels vs Left Ventricular Dysfunction

- 64% of our study population had LV dysfunction.
- The association between the vitamin D levels and LV dysfunction was not significant.(p=0.21) . this could be due to the fact that many of our patients were effort angina patients who would not have sustained myocardial damage in the absence of MI to produce the LV dysfunction

vitamin D levels vs MR

- Our study did not show any association between vitamin D levels and MR (p=0.12). this could be due to other confounding factors such as age, high prevalence of physiological MR, and the frequency to which MI patients especially inferior wall MI patients have MR, irrespective of angiographic severity or vitamin D status.

vitamin D levels vs aortic sclerosis

- Our study did not show any association between vitamin D levels and the presence of aortic sclerosis. (P=0.18)
- This could be due to other confounding factors such as age which commonly is associated with aortic sclerosis.

vitamin D levels vs Diastolic Dysfunction

- 87% of our study population had diastolic dysfunction of varying grades.
- The association between the vitamin D levels and diastolic dysfunction was **significant**(P=0.000)
- This could reflect the fact that lower vitamin D levels cause greater degrees of hypertrophy and stiffening of LV walls reducing their compliance as vitamin D has been shown to regulate myocardial hypertrophy[81].

vitamin D levels vs normal coronaries

- 12% of our subjects had normal coronaries in the angiogram
- The association between the vitamin D levels and normal coronaries was **significant**(P=0.02)
- the average vitamin D levels in those with normal coronaries was 24.2 ng/ml. This is significantly higher than in that of the entire study population but still in the hypovitaminosis range.

- Of those with normal coronaries only 15% had a vitamin D value of less than 20ng/ml.61% had vitamin D levels in the hypovitaminosis range (20-30ng/ml).
- Hence we see that patients with normal coronaries tended to have a higher mean vitamin D level though still in the below adequate levels.

Vitamin D levels vs Single vessel disease

- 31% of our subjects had single vessel disease.
- The association between the vitamin D levels and SVD was not significant. (P=0.07)
- The average vitamin D levelsin SVD patients was 22.6 ng/ml.
- On analyzing the vitamin D level subgroups in SVD patients, 23% had adequate vitamin D (>30ng/ml), 38% had vitamin D levels in the hypovitaminosis range (20-30ng/ml) and 29% had vitamin D levels in the insufficient range (10-20ng/ml) and only 8% had <10ng/ml. We see that a patient with SVD was less likely to have a vitamin D level of <10 ng/ml even though statistical significance was not reached.

vitamin D levels vs double vessel disease

- 25% of our study population had DVD on angiograms.
- The association between the vitamin D levels and DVD was not significant. (P=0.19)
- The average vitamin D levels in DVD patients was 18.3ng/ml. Though statistical significance was not reached the vitamin D levels in DVD patients was lower than the mean levels in SVD and those with normal coronaries, showing a gradation that lower mean vitamin D levels are encountered in patients with more number of coronaries involved.

vitamin D levels vs triple vessel disease

- 31% of our subjects had TVD on angiograms.
- The association between the vitamin D levels and TVD was **significant**(P=0.000)
- The average vitamin D levels in TVD patients was 13.8ng/ml , significantly lower than that seen in the entire study subjects.
- On analyzing the vitamin D level subgroups in TVD patients, only 5% had adequate vitamin D (>30ng/ml), 8% had

vitamin D levels in the hypovitaminosis range (20-30ng/ml) and 52% had vitamin D levels in the insufficient range (10-20ng/ml) and 38% had <10ng/ml. Almost 90% of those with TVD had vitamin D levels <20ng/ml

- Of the total subjects who had a vitamin D level of <10 ng/ml, 68% had a triple vessel disease.

vitamin D levels vs Lt main involvement

- 11% of our subjects had lt main art involved .
- The association between the vitamin D levels and Lt main inv was **significant**(P=0.01)
- average vitamin D levels in lt main patients was 11.9ng/ml ,
- None of those with left main inv had adequate vitamin D levels (>30ng/ml), 8% had vitamin D levels in the hypovitaminosis range (20-30ng/ml) and 41% had vitamin D levels in the insufficient range (10-20ng/ml) and 50% had <10ng/ml.
- Of the total subjects who had a vitamin D level of <10 ng/ml, 31% had a lt main inv with or without involvement of other arteries.

- It is evident that lt main inv is more common as vitamin D levels falls and is most common in those with the lowest levels. The average vitamin D level was significantly lower than in other arterial involvement.

Vitamin D levels vs 100% cutoff

- 21% of our subjects had 100% cut of f in coronary angiograms.
- The association between the vitamin D levels and 100% cut off was **significant**(P=0.000)
- of those with 100% cutoff , only 4% had adequate vitamin D levels (>30ng/ml), 8% had vitamin D levels in the hypovitaminosis range (20-30ng/ml) and 43% had vitamin D levels in the insufficient range (10-20ng/ml) and 43% had <10ng/ml.
- Of the total subjects who had a vitamin D level of <10 ng/ml, 52% had a 100% cutoff.
- Hence we see that as vitamin D levels fall, the incidence of 100% cut off increases and below 20ng/ml the incidence is extremely significant.

Vitamin D levels vs Gensini score

- The association between vitamin D levels and Gensini score was significant (p=0.000)
- The mean Gensini score for the entire study population was 38.84 ± 29.85.
- Of those with adequate vitamin D levels (>30ng/ml), the mean Gensini score was 21.25 ± 20.06, in vitamin D levels in the hypovitaminosis range (20-30ng/ml) the mean Gensini score was 25.42 ± 23.97 and in the insufficient range (10-20ng/ml) the mean Gensini score was 42.50 ± 25.42, in those with <10ng/ml the mean Gensini score was 62.00 ± 35.83.
- It is seen that as vitamin D levels fall in the population there is a graded increase in the Gensini score with the highest Gensini scores seen in the lowest vitamin D levels.

vitamin D levels vs ABI

- The association between vitamin D levels and ABI was not significant (p=0.37)

vitamin D levels vs eGFR

- The association between vitamin D levels and eGFR was not significant (p=0.33)

STUDY LIMITATIONS

The study was conducted in a single center.

The sample size of the study group was small. Both these factors could have introduced a selection bias.

The inclusion criteria was patients undergoing coronary angiogram, the vitamin D levels in those who do not undergo angiograms were not tested. Hence these findings cannot be extrapolated to the general population.

The long term event rates in these patients was not assessed as there was no long term followup.

CONCLUSION

This study included 108 patients who underwent coronary angiogram for various indications such as acute coronary syndromes, effort angina/chronic stable angina and for other diagnostic indications.

The mean age of our population was 52.41

The prevalence of vitamin D deficiency (<30ng/ml) in the entire study population was 86%.

The mean vitamin D level in the entire study population was insufficient (19.00±8.42) ng/ml.

This is similar to other angiographic studies conducted on Indian patients [98,99,100]. The prevalence of vitamin D deficiency in patients undergoing angiogram in the study by Sanjeev Kumar Syal et al[100] was 80%. Hence the high prevalence of inadequate vitamin D in the patient population undergoing coronary angiogram was reinforced in our study raising the possibility of vitamin D supplementation in this high risk group.

The prevalence of vitamin D deficiency (<30ng/ml) in the ACS subgroup was 86% and in the EA group was 92%.

There was a graded relationship between lower quartiles of vitamin D levels and the proportion of patients having ACS .

Of the study group with the lowest quartile of vitamin D levels(<10ng/ml) the indication for angiogram was ACS in 84% suggesting that this subgroup has a significantly high risk for developing an acute coronary syndrome.

There was no association between the vitamin D levels and many demographic factors such as age, sex, smoking, diabetes, hypertension and obesity. The lack of association is probably due to the invariably high prevalence of vitamin D deficiency in this population irrespective of the demographic factors.

There was an association between vitamin D levels and hard clinical endpoints and in hospital events such as reinfarction, death, ionotropic support, arrhythmias, presence of diastolic dysfunction and the presentation of the patients such as ACS or chronic stable angina /EA, and the presence of normal coronaries, triple vessel disease, lt main involvement and 100% cutoff in the coronary angiogram. The p value was significant in all these parameters, thereby suggesting that lower vitamin D levels significantly affect the extent of atherosclerotic burden.

Angiographically severe CAD as manifested by TVD, Lt main involvement and 100% cutoff was significantly associated with lower vitamin D levels in a graded manner and most evident in those with the lowest quartile of vitamin D levels(<10ng/ml) thereby putting these patients at high risk of mortality and the need for CABG.

The Gensini score is a well validated measure of assessing the severity of angiographic lesions[116]. The mean Gensini score for those with adequate vitamin D levels was 21.25 . This score increased progressively with lower vitamin D levels and in the lowest quartile of vitamin D (<10ng/ml) the mean Gensini score was 62.00 .

The association between vitamin D levels and the Gensini score was significant showing that lower vitamin D levels had a higher Gensini score and hence greater severity of atherosclerotic lesions.

In conclusion it was shown that vitamin D deficiency was highly prevalent in the population subgroup undergoing angiogram and those with lower vitamin D levels were more likely to develop an Acute Coronary Syndrome and more likely to have reinfarction, death, require ionotropic support and arrhythmias and more likely to have severe extent of atherosclerosis such as triple vessel disease and left main involvement and 100% cutoff with higher Gensini scores.

Thus the possibility that vitamin D status can be added to the classical risk factor profiles and vitamin D supplementation could be used as a new mode of risk factor modification in this high risk population is an intriguing prospect.

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INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work	: Prevalence of Vitamin D deficiency in CAD patients (ACS and stable angina) and correlation with Angiographic severity
Principal Investigator	: Dr.A. Arvind
Designation	: PG in D.M (Cardiology)
Department	: Department of Cardiology Government Stanley Medical College, Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 07.02.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

PREVALENCE OF VITAMIN D DEFICIENCY IN CAD PATIENTS (ACS AND STABLE ANGINA) AND CORRELATION WITH ANGIOGRAPHIC SEVERITY

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E-mail	aviasokkumar@yahoo.com
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1 INTRODUCTION Coronary artery disease (CAD),is a leading cause of mortality and morbidity world wide and has reached epidemic proportions. Ischemic heart disease causes 9.4% of total deaths (2.5 million) in less developed countries and 16.3% (1.3 million) of all deaths in well developed countries1. The WHO has estimated that in the year 2002 alone, 12.6% of deaths all over the world were due to coronary artery disease2. The proportion of these diseases of aging is expected to increase as the world population gets older. The Indian scenario is similar. Studies have shown that cardiovascular diseases cause about 40% of deaths in the urban centers and 30% of deaths in rural centers in our...

PROFORMA

Name: **Age:** **Sex:**

Occupation: **DOA:**

Address: **IP NO:**

CD NO:

Diagnosis:

Risk Factors:

Male Gender: Diabetes:

Smoker: Dyslipidemia

Hypertension: Family History of CAD:

In Hospital Therapy: Thrombolysis:

Inotropic Therapy:

On Examination:

HT: WT: BMI: BSA:

PR: BP:

CVS: RS:

ECG:

Killip Class On Admission:

NYHA class for CSA patients:

Investigations:

Complete Blood Count:

Urea: Creatinine: eGFR :

Electrolytes: Lipids: ABI:

CPK/MB:

VITAMIN D LEVELS:

ECHO:

LVEF, %:

$LVID_D$, mm: $LVID_S$, mm:

Diastolic Function:

MR : PHT : Aortic sclerosis :

In Hospital Events:

Death: Re-MI:

Arrhythmias:

LV Dysfunction: Mechanical Complication: VSR / MR

Coronary angiogram:

Gensini score :

CONSENT FORM

I agree to participate in the study titled - “PREVALENCE OF VITAMIN D DEFICIENCY IN CAD PATIENTS (ACS AND STABLE ANGINA) AND CORRELATION WITH ANGIOGRAPHIC SEVERITY”.

I confirm that I have been told about this study in my mother tongue and have had the opportunity to ask question.

I understand that my participation is voluntary and I may refuse to participate at any time without giving any reason and without affecting my benefits.

I agree not to restrict the use of any data or results that arise from the study.

I agree to undergo the necessary investigation which is part of the study.

Name of the participant:

Signature / thumb impression

Investigator

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

இருதய மாரடைப்பு நோயாளிகளில்
வைட்டமின் டி குறைபாட்டின் பரவல் சதவீதமும், இருதயத் தமனிகளின்
அடைப்பின் தீவிரத்தையும் பற்றிய ஒப்பீட்டு ஆய்வு,

ஆராய்ச்சி நிலையம் : அரசு ஸ்டான்லி மருத்துவமனை
சென்னை - 600 001.

பங்கு பெறும் நோயாளியின் பெயர் : வயது :

பங்கு பெறும் நோயாளியின் எண் : பாலினம் : ஆண் பெண்

நோயாளியின் விலாசம்

நோயாளி இதனை ( ) குறிக்கவும்.

மேலே குறிப்பிடப்பட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும். அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. [ ]

நான் என்னை இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்க அனுமதிக்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் என்னை இவ்வாய்வில் இருந்து விலக்கி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். [ ]

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். என்னை ஆய்வில் இருந்து விலக்கிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். [ ]

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். [ ]

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணிக்கு தெரிவிப்பேன் என உறுதி அளிக்கிறேன். [ ]

நோயாளியின் கையொப்பம் ..... இடம் .....தேதி

கட்டைவிரல் ரேகை (இந்த படிவம் படித்து காட்டப்பட்டு புரிந்து கைரேகை அளிக்கின்றேன்)

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

.....

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளரின் பெயர்.....

S.NO	NAME	AGE	SEX	DIABETES	HYPERTENS	SMOKING	DYSLIPIDEM	HEIGHT (cm)	WEIGHT(kg)	BMI kg/	DIAGNOSIS	NYHA	KILLIP	INOTROPIC	THROMBOI	ARRYTHMIA	EF %	aortic scler	PHT	MR	DIASTOLIC	VIT D ng/m	eGFR ml/hı	ABI	normal cor	SVD	DVD	TVD	100% cut o	LT MAIN	Gensini scc	RE MI	DEATH
	1 rahib khan		62 m	-	-	-	+	182	91	27.5	IMMI+PWMI+RVMI	-	1	-	+	-	45	+	+	mild	1	34	108	1	-	-	-	+	-	-	16	-	-
	2 abdul khader		60 m	-	-	+	+	160	60	23.4	IWMI+RVMI	-	1	-	+	-	45	+	-	mild	1	18.8	55.56	1	-	-	-	+	+	-	61	-	-
	3 krishnakumari		52 f	-	+	-	-	154	69	29.1	ASMI-2008/EA	2	-	-	-	-	60	-	-	mild	2	8.8	140	1	-	+	-	-	-	-	3	-	-
	4 ahamed sharif		50 m	-	+	+	+	170	70	24.2	EXT AWMI/EA	3	1	-	+	RBBB+LAFB	32	-	-	mild	2	9.8	145	0.98	-	-	-	+	+	-	96	-	-
	5 tamil malai		41 m	-	-	+	+	164	59	23	AWMI 2009/EA	3	-	-	-	-	57	-	-	-	3	21	100	1.03	-	-	+	-	+	-	108	-	-
	6 sarojini		65 f	+	-	-	+	148	61	27.8	NSTEMI/EA	3	-	-	-	-	64	+	-	mild	1	18.4	99	1.01	-	-	-	+	-	+	54	-	-
	7 chandrababu		47 m	-	+	+	+	159	57	22.5	EA	3	-	-	-	-	70	+	-	mild	1	11.9	81	1.03	-	-	-	+	+	-	68	-	-
	8 ashok		48 m	-	+	+	-	170	75	26	IWMI+PWMI	-	1	-	-	-	40	-	-	-		26	95	1.1	+	-	-	-	-	-	-	-	-
	9 rupavathy		53 f	-	+	-	-	147	59	27	EA	3	-	-	-	-	64			mild	1	12.7	89	1	-	-	+	-	+	-	43	-	-
	10 anbalagan		55 m	+	+	+	+	165	64	23.5	IWMI+PWMI	-	2	+	+	CHB	45	+	mild	mild	3	15.8	75	1.1	-	+	-	-	-	-	2	-	-
	11 panneer selvam		70 m	+	-	+	+	161	70	27	Ext AWMI		2	+	+	-	40	+	-	-	2	18.1	88	0.9	-	+	-	-	-	-	24	-	-
	12 govindaraj		56 m	-	-	-	+	172	71	24	AWMI	-	3	+	-	-	42	+	mild	-	2	8.57	103	0.99	-	-	-	+	+	-	58	-	-
	13 rajendran		52 m	+	-	+	-	166	65	23.6	IWMI+RVMI	-	1	-	-	-	45	-	-	-	2	8.49	88.27	1.02	-	-	-	+	-	+	41	-	-
	14 kennedy		50 m	-	-	+	+	160	60	23.4	IWMI+PWMI+RVMI	-	2	-	+	1st deg AV bl	52	-	-	mild	1	22.2	83	1.02	-	+	-	-	-	-	25	-	-
	15 chinnaiah		68 m	+	+	-	-	166	75	27.2	EA	3	-	-	-	-	66	+	-	-	1	28	125	1	-	-	-	+	-	-	52	-	-
	16 suresh		43 m	-	-	+	+	165	72	26.4	AWMI	-	3	+	+	-	40	-	-	mild	1	25	121	1	+	-	-	-	-	-	-	-	-
	17 dawood basha		52 m	-	-	-	-	163	66	24.8	IWMI+PWMI+LWMI	-	1	-	+	-	59	-	-	-	-	14.8	73	1	-	-	-	+	+	-	59	-	-
	18 ashok		42 m	-	+	+	+	164	64	23.8	IWMI+PWMI	-	1	-	-	1st deg AV bl	40	-	-	-	1	15	67	0.9	+	-	-	-	-	-	-	-	-
	19 manavalan		54 m	-	-	-	-	165	65	23.9	IWMI+PWMI+RVMI	-	1	-	+	2:1 AV block	45	+	-	mild	1	12.2	110	1	-	+	-	-	-	-	8	-	-
	20 kathirvel		43 m	-	+	+	+	159	78	30.9	UA	-	-	-	-	-	60	-	-	-	-	12.9	150	1	-	-	-	+	-	-	70	-	-
	21 kanagavalli		54 f	+	+	-	-	156	70	28.8	NSTEMI	-	-	-	-	RBBB	45	+	-	mild	1	6.51	92	0.98	-	-	-	+	+	+	164	+	-
	22 malathy		48 f	-	-	-	-	146	53	24.9	HLMI	-	1	-	+	-	47	+	-	mild	3	8.01	96	1.12	-	+	-	-	-	-	28	-	-
	23 kennedy		43 m	-	-	+	-	175	69	22.5	IWMI+PWMI+RVMI	-	1	-	+	-	60	+	-	mild	-	30	80	1.1	-	-	+	-	-	-	5	-	-
	24 mohamad rafiq		40 m	-	-	+	+	169	101	35	IWMI+PWMI	-	1	-	+	1st deg AV bl	45	-	-	mild	1	8.9	154	0.97	+	-	-	-	-	-	-	-	-	-
	25 rajendran		60 m	-	-	-	+	157	66	26.8	IWMI	-	1		+	CHB	52	+	-	mild	1	8.8	73	0.89	-	-	-	+	+	-	63	-	-
	26 lathif		50 m	-	-	+	-	157	52	21.1	IWMI	-	1	-	+	1st deg AV bl	60	+	-	-	1	13.7	72	0.98	-	+	-	-	-	-	10	-	-
	27 jahangir basha		62 m	-	-	+	+	162	76	29	EA	2	-	-	-	-	49	+	-	mild	1	12.3	64	0.89	-	+	-	-	+	-	80	-	-
	28 govindaraj		57 m	-	-	-	-	164	63	23.4	IWMI	-	1	-	+	-	57	-	-	-	-	22.8	98	1	-	-	-	+	-	-	59	+	-
	29 abdul rahman		35 m	-	-	+	-	160	60	23.4	AWMI	-	1	-	+	-	45	-	-	-	1	13	96	1	-	-	+	-	-	-	16	-	-
	30 selvam		47 m	+	+	+	+	172	66	22.3	EA	3	-	-	-	-	56	-	+	mild	1	12.7	66	1.12	-	-	-	+	-	-	48	-	-
	31 shabana begum		37 f	-	-	-	+	150	65	28.9	EA	2	-	-	-	-	60	-	-	-	-	22	76	1.1	+	-	-	-	-	-	-	-	-
	32 jeeva		49 m	+	-	+	-	166	70	25.4	old ASMI/recurrent UA	-	-	-	-	-	48	-	-	mild	1	22.1	61	1	-	+	-	-	-	-	20	-	-
	33 sultana begum		52 f	+	+	-	+	149	60	27	NSTEMI	-	-	-	-	-	47	-	+	mild	1	14	65	1.01	-	-	+	-	-	-	57	-	-
	34 chandra		63 f	+	-	-	+	152	53	22.9	EA	2	-	-	-	-	61	+	+	moderat	1	23.1	60	1	-	+	-	-	-	-	28	-	-
	35 pari		50 m	-	+	+	-	170	68	23.5	EA	3	-	-	-	-	57	+	+	mild	3	11	59	1	-	-	-	+	-	-	38	-	-
	36 arokiathavaman		43 m	+	-	+	-	171	78	24.6	old AWMI/EA	2	-	-	-	-	44	-	-	-	2	28	78	1	-	-	+	-	-	-	19	-	-
	37 Karuppanan		64 m	+	+	-	-	161	76	29.3	old IWMI/EA	3	-	-	-	-	49	+	+	mild	3	10.1	64	1	-	-	-	+	-	-	36	-	-
	38 saravanan		32 m	-	-	+	-	168	71	25.2	AWMI	-	1	-	+	-	46	-	-	-	1	21.2	87	1.03	-	+	-	-	-	-	20	-	-
	39 srinivasan		36 m	-	-	+	+	173	78	26.1	AWMI	-	1	-	+	-	50	-	-	-	1	18	98	1.2	-	+	-	-	-	-	12	-	-
	40 antony dasan		55 m	+	-	+	+	175	82	26.8	EA	2	-	-	-	LBBB	57	+	-	-	2	8	77	1.02	-	-	-	+	+	-	94	-	-
	41 meenakshi		52 f	+	+	-	+	156	70	28.8	EA	2	-	-	-	-	60	-	+	-	2	28	95	1	-	-	+	-	-	-	14	-	-
	42 rajasekhar		46 m	-	+	+	-	178	92	29	AWMI	-	3	+	+	-	40	-	+	mild	3	12.1	87	1	-	-	-	+	-	-	16	-	-
	43 sundar		52 m	+	+	-	-	174	81	26.8	IWMI+PWMI+RVMI	-	1	-	+	1 deg AV bloc	45	-	-	mild	1	10.2	80	1	-	-	-	+	-	-	26	-	-
	44 john basha		53 m	-	-	+	-	165	82	30.1	NSTEMI	-	-	-	-	-	51	-	-	-	1	34	100	1.1	-	+	-	-	-	-	2	-	-
	45 venkateswarulu		64 m	+	-	-	+	173	88	29.4	IWMI+PWMI	-	1	-	+	1 deg AV bloc	50	+	+	mild	1	9.7	86	0.9	-	-	-	+	-	-	34	-	-
	46 alagammal		52 f	+	-	-	-	150	60	26.7	EA	3	-	-	-	-	60	-	-	-	1	13	91	1	-	-	-	+	-	-	44	-	-
	47 nagarajan		36 m	-	+	+	-	171	80	27.4	Ext AWMI	-	1	-	+	-	50	-	-	-	1	29.1	101	1	-	+	-	-	-	-	2	-	-
	48 narasimman		67 m	+	+	-	+	175	79	25.8	EA/AS/AR	2	-	-	-	-	52	+	+	mild	1	11	63	0.9	-	-	-	+	-	-	50	-	-
	49 abdul latif		60 m	+	+	-	+	160	75	29.3	IWMI	-	1	-	-	CHB	50	+	-	mild	1	8.8	76	1	-	-	-	+	-	+	77	+	-
	50 ayyadurai		60 m	-	+	-	+	177	82	26.2	renal donor	-	-	-	-	-	66	-	-	-	-	32	98	1.02	+	-	-	-	-	-	-	-	-
	51 durai		42 m	+	-	+	-	174	88	29.1	Ext AWMI	-	1	-	+	-	53	-	-	-	1	35	121	1.03	+	-	-	-	-	-	-	-	-
	52 janarthanan		62 m	+	-	+	-	163	75	28.2	EA	2	-	-	-	-	58	+	-	mild	1	30	98	1	-	-	-	+	-	-	65	-	-
	53 periyasamy		65 m	-	+	+	+	169	84	29.4	old IWMI /EA	-	-	-	-	-	54	+	+	mild	1	25	113	1	+	-	-	-	-	-	-	-	-

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63	lakshmanasamy	75	m	+	+	-	+	162	78	27.6	IWMI	-		3	+	+	mobitz 2	48	+	+	mild		1	15.3	68	0.89	-	-	+	-	-	-		52	-	-
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71	chandra	60	m	+	-	-	-	161	70	27	EA/AS		2	-	-	-	-	57	+	+	mild		3	26.8	85	1.11	-	+	-	-	-	-		5	-	-
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E-mail	aviasokkumar@yahoo.com
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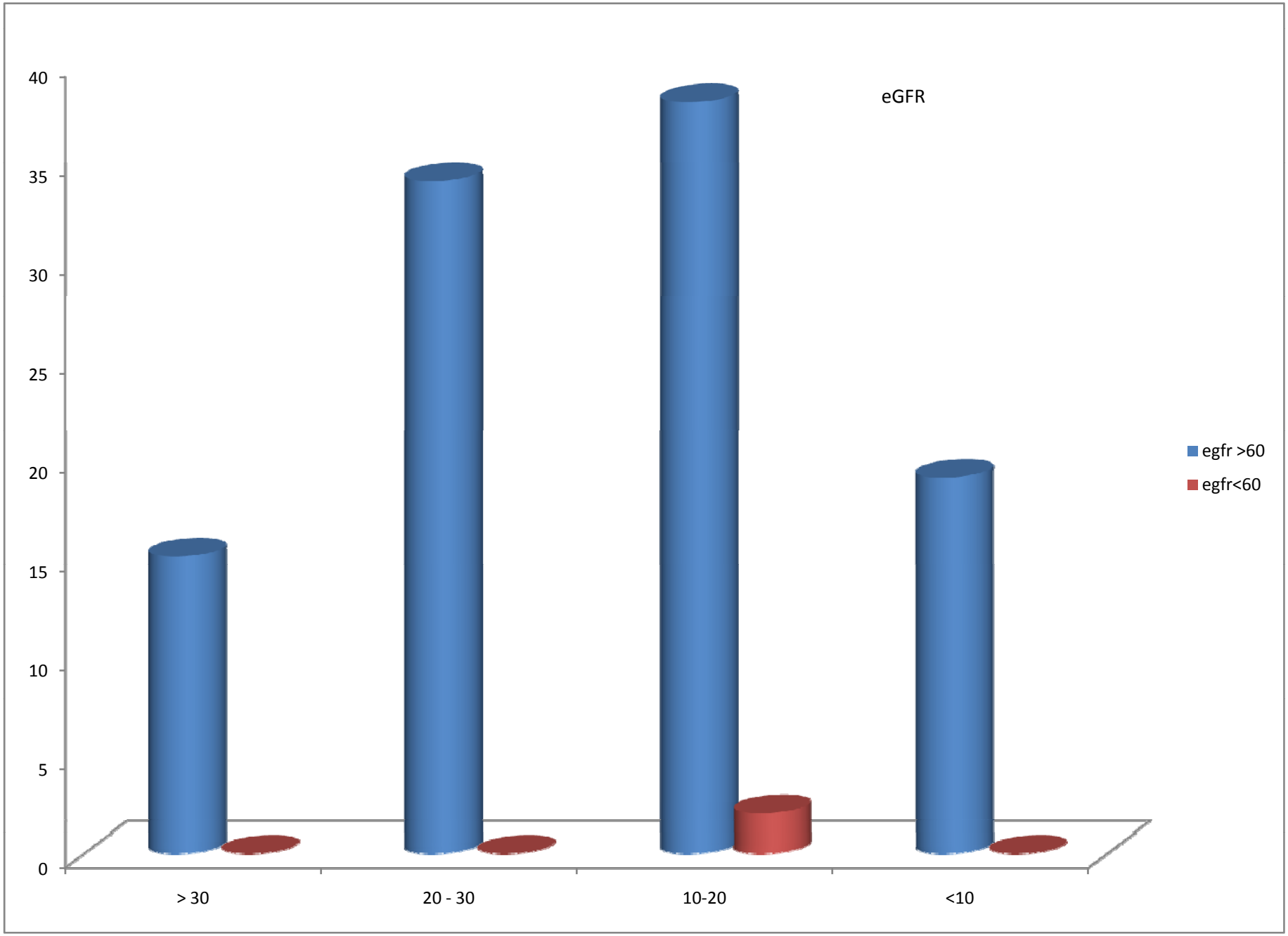
1 INTRODUCTION Coronary artery disease (CAD),is a leading cause of mortality and morbidity world wide and has reached epidemic proportions. Ischemic heart disease causes 9.4% of total deaths (2.5 million) in less developed countries and 16.3% (1.3 million) of all deaths in well developed countries1. The WHO has estimated that in the year 2002 alone, 12.6% of deaths all over the world were due to coronary artery disease2. The proportion of these diseases of aging is expected to increase as the world population gets older. The Indian scenario is similar. Studies have shown that cardiovascular diseases cause about 40% of deaths in the urban centers and 30% of deaths in rural centers in our...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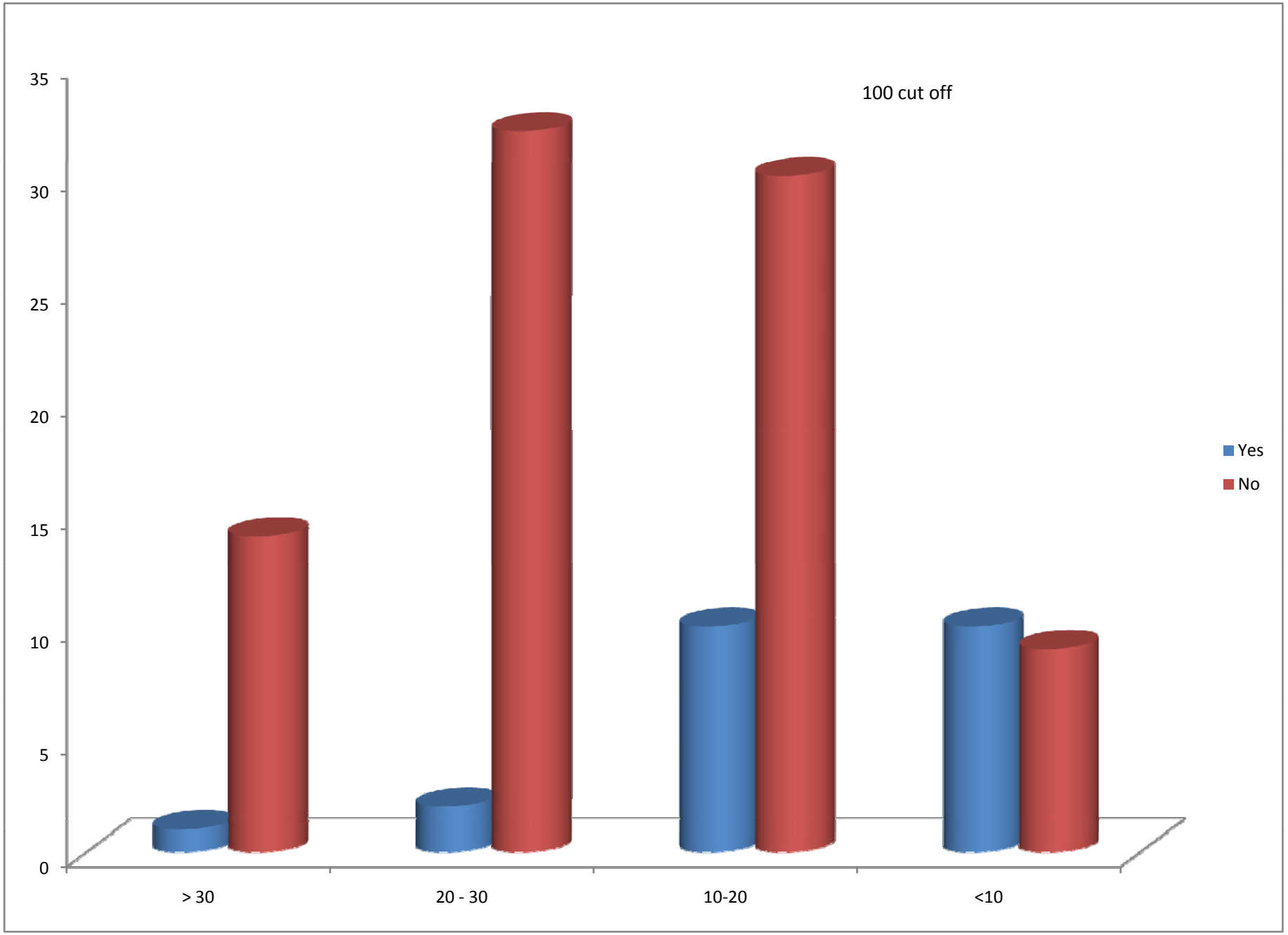
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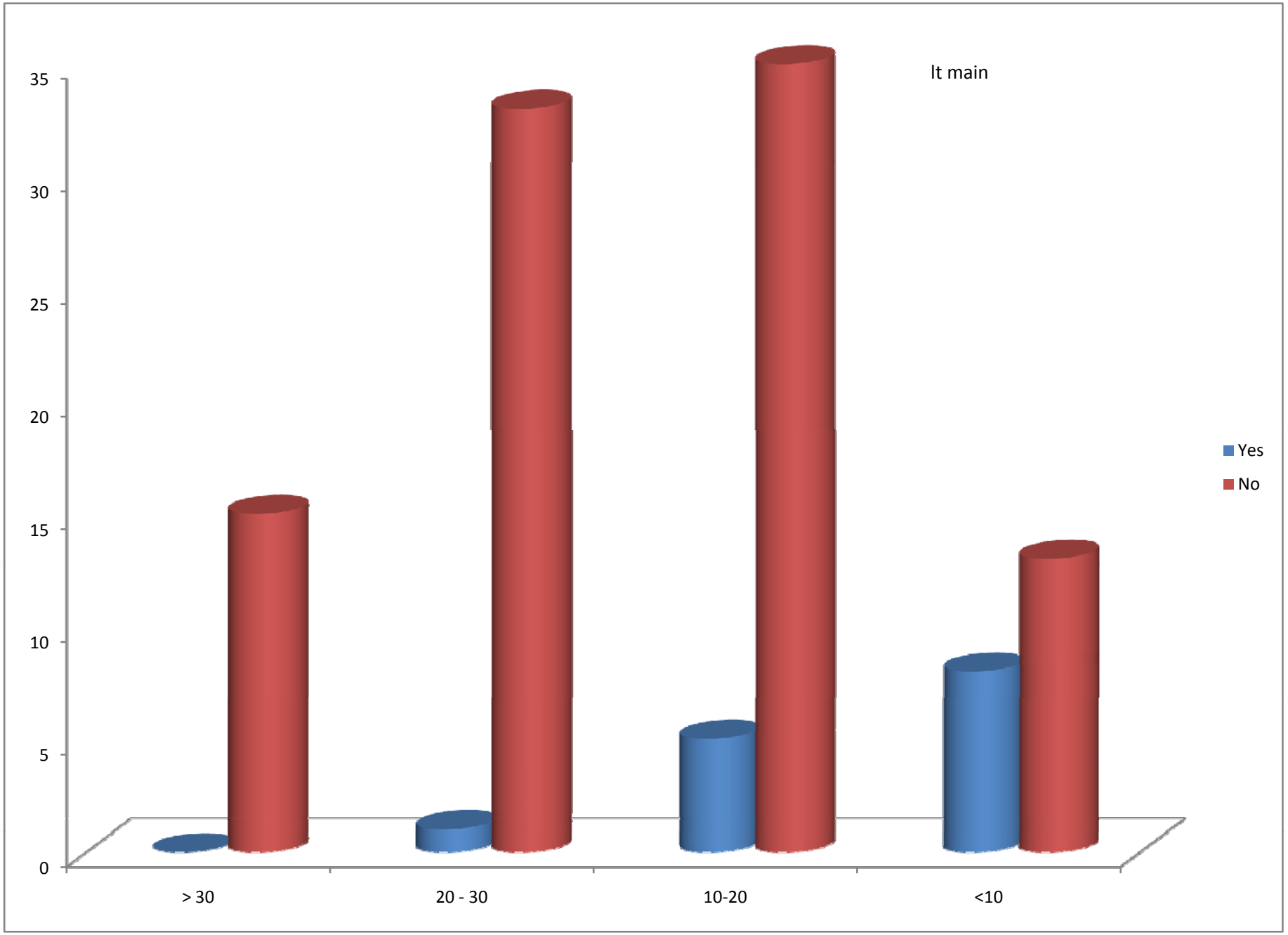
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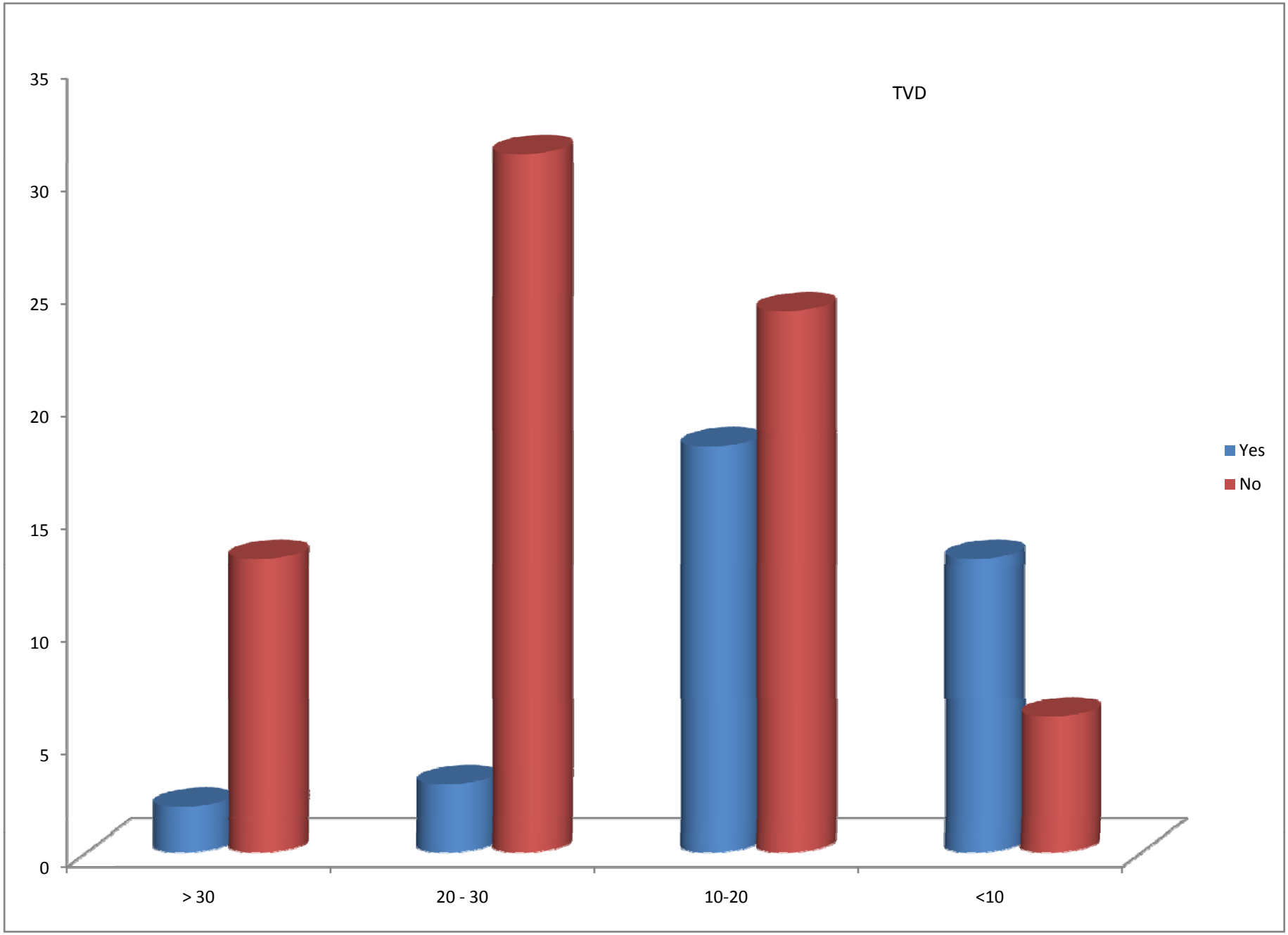
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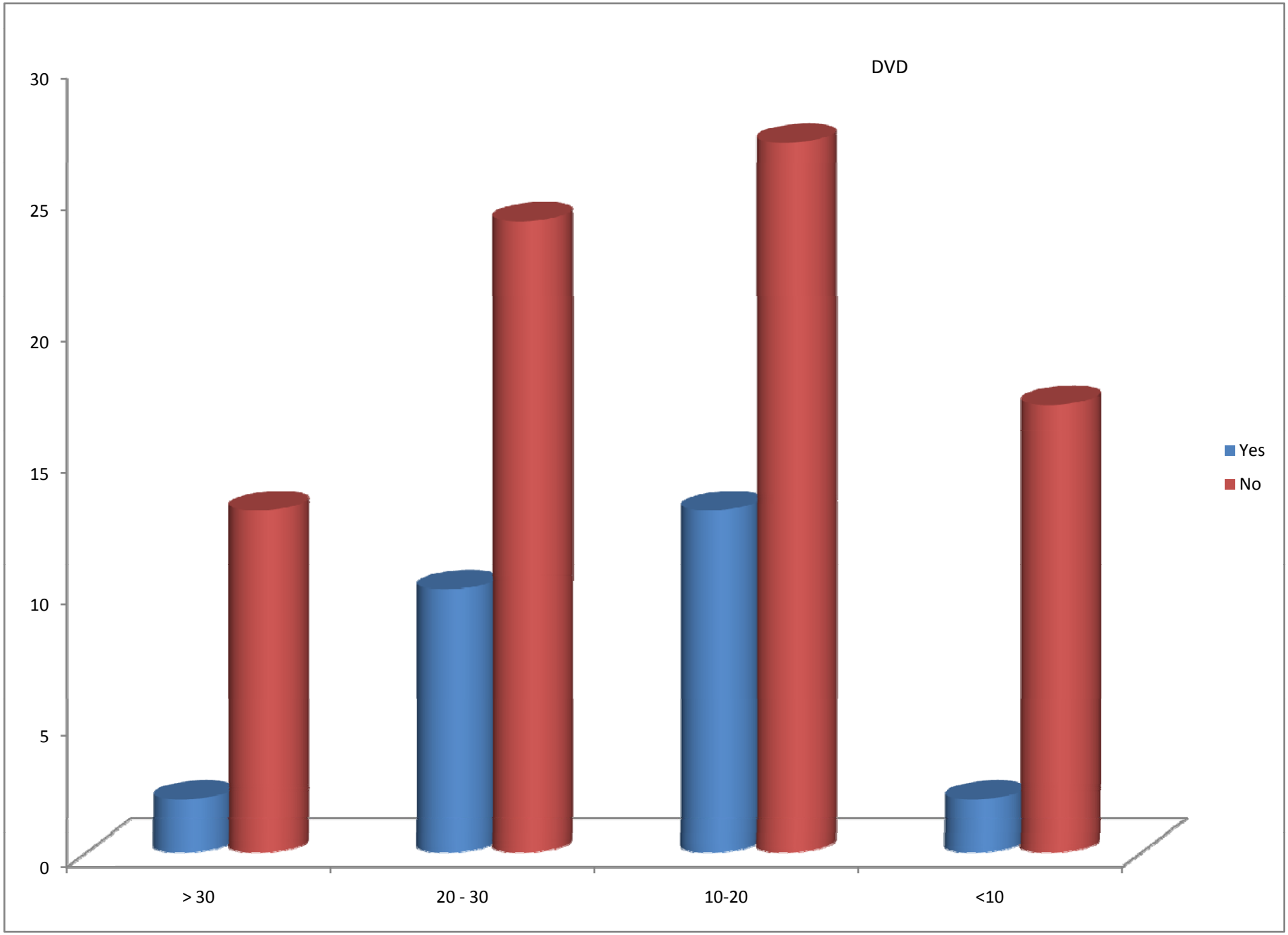
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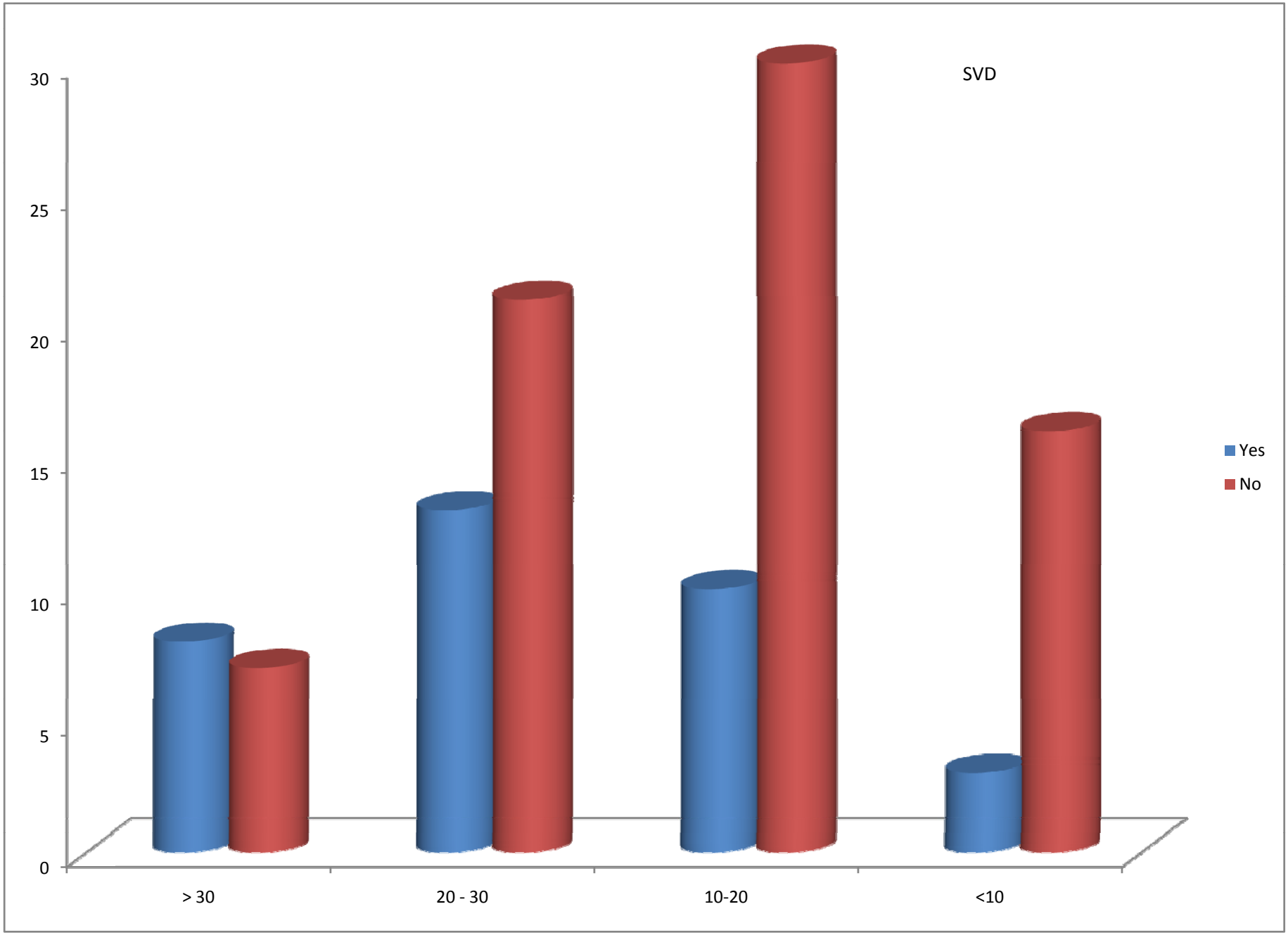
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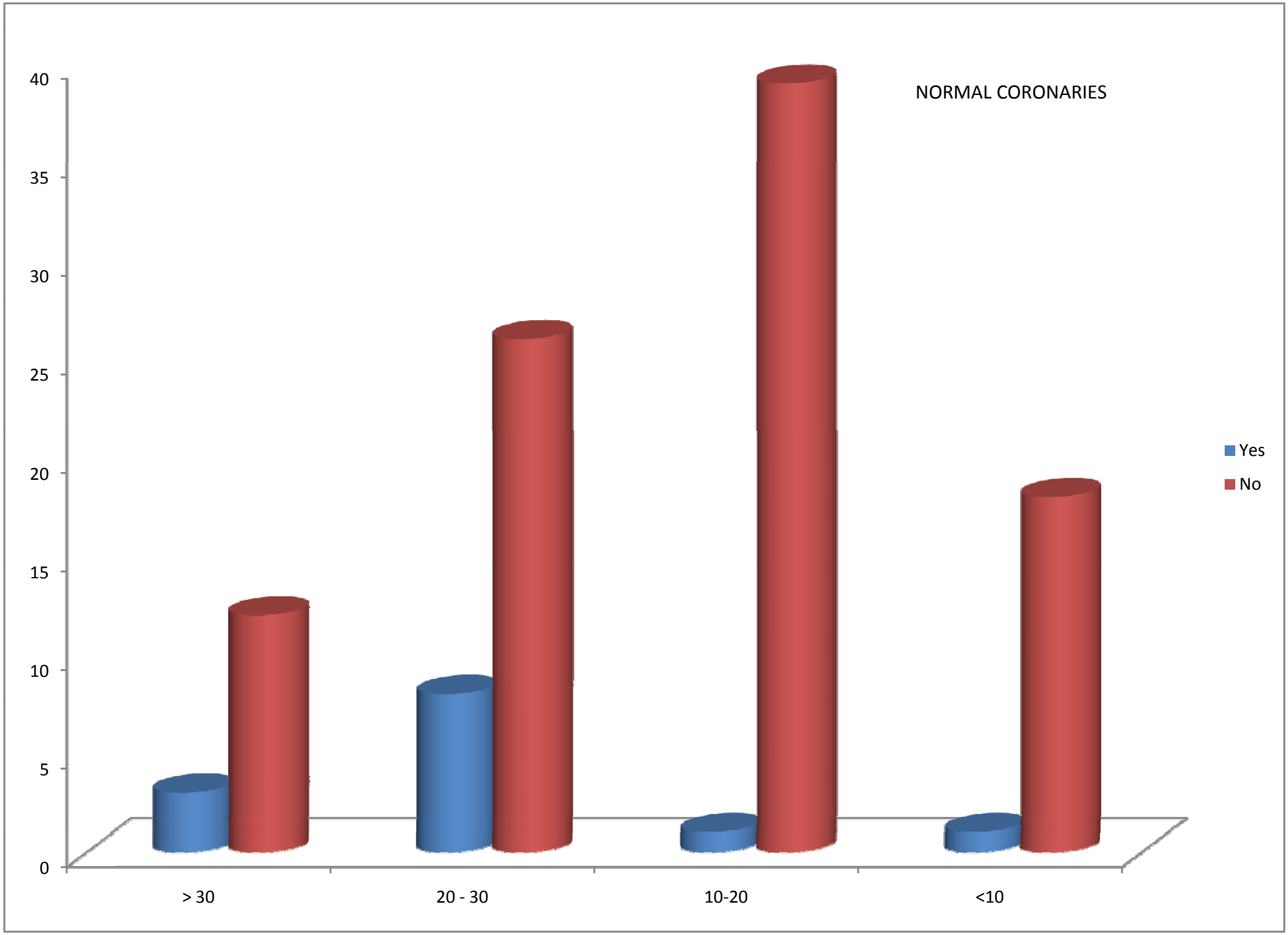
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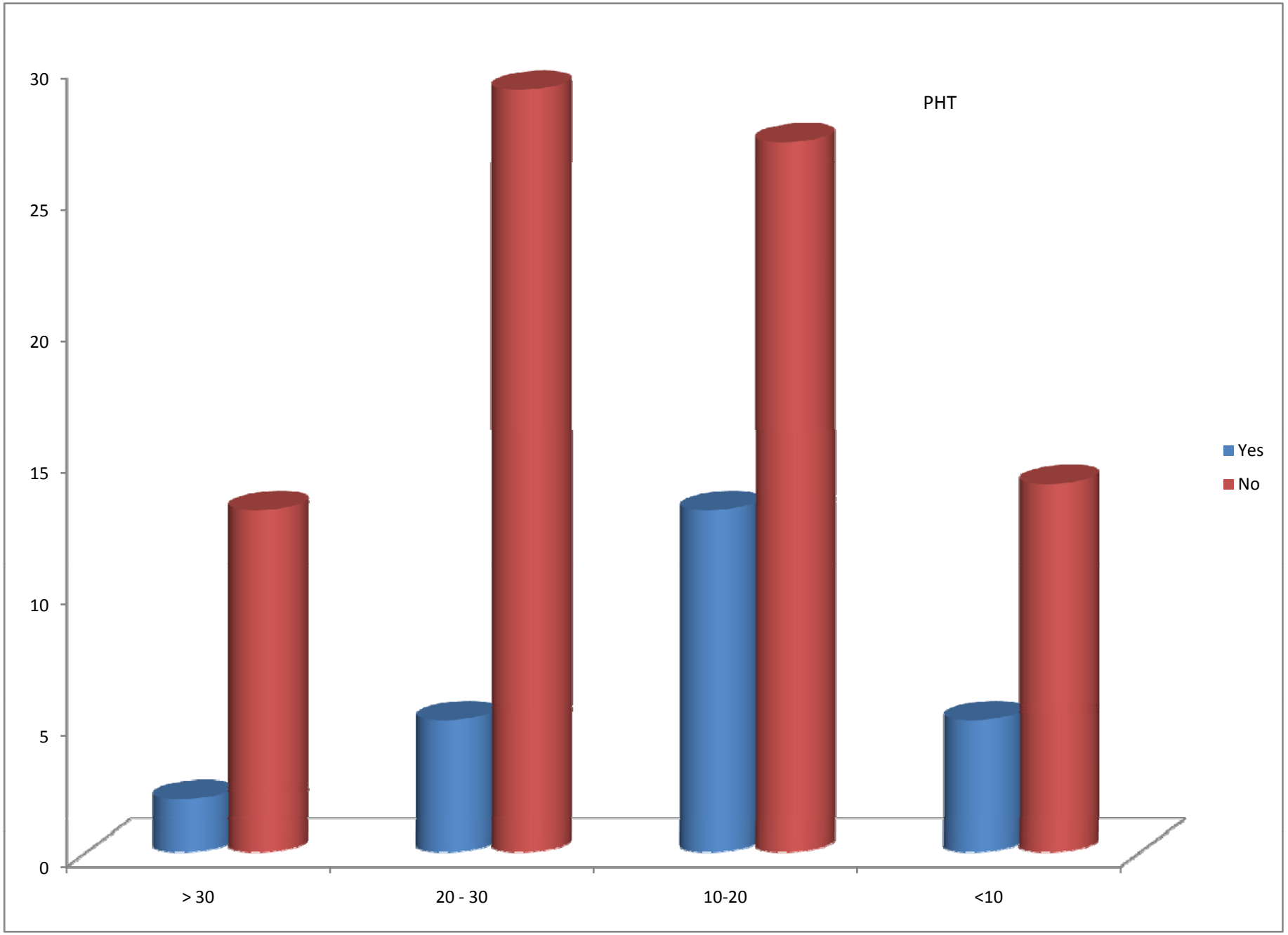
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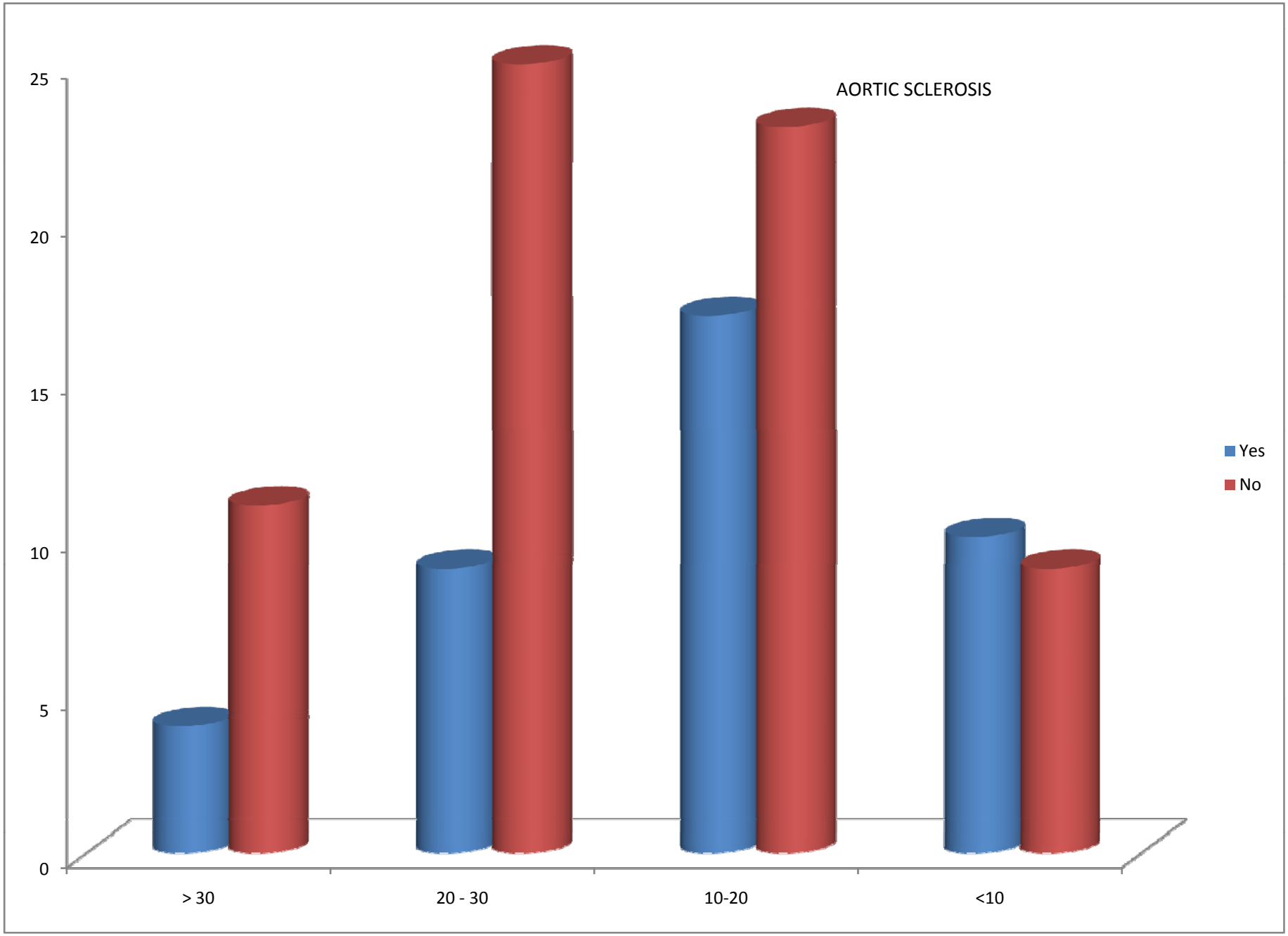
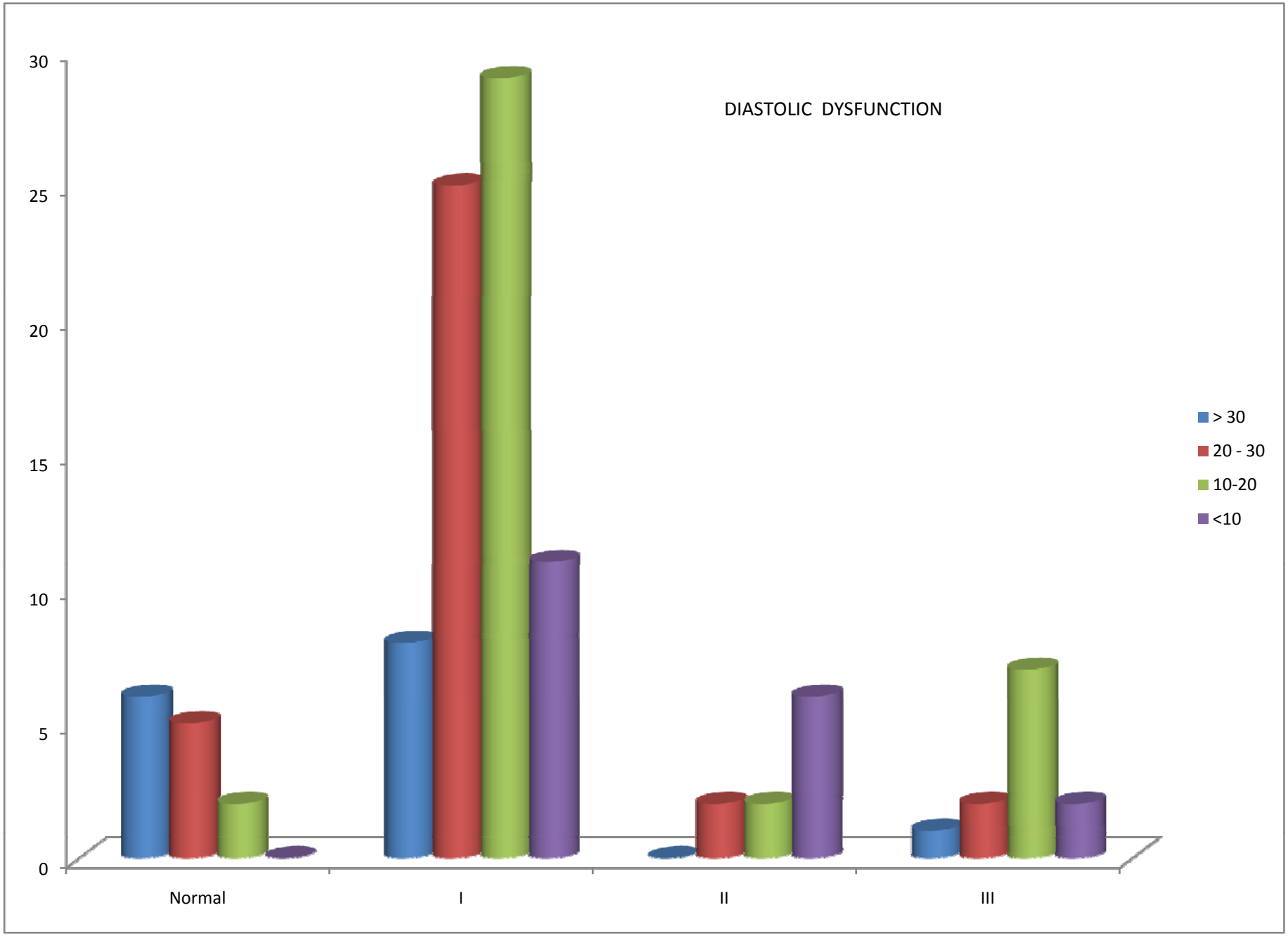
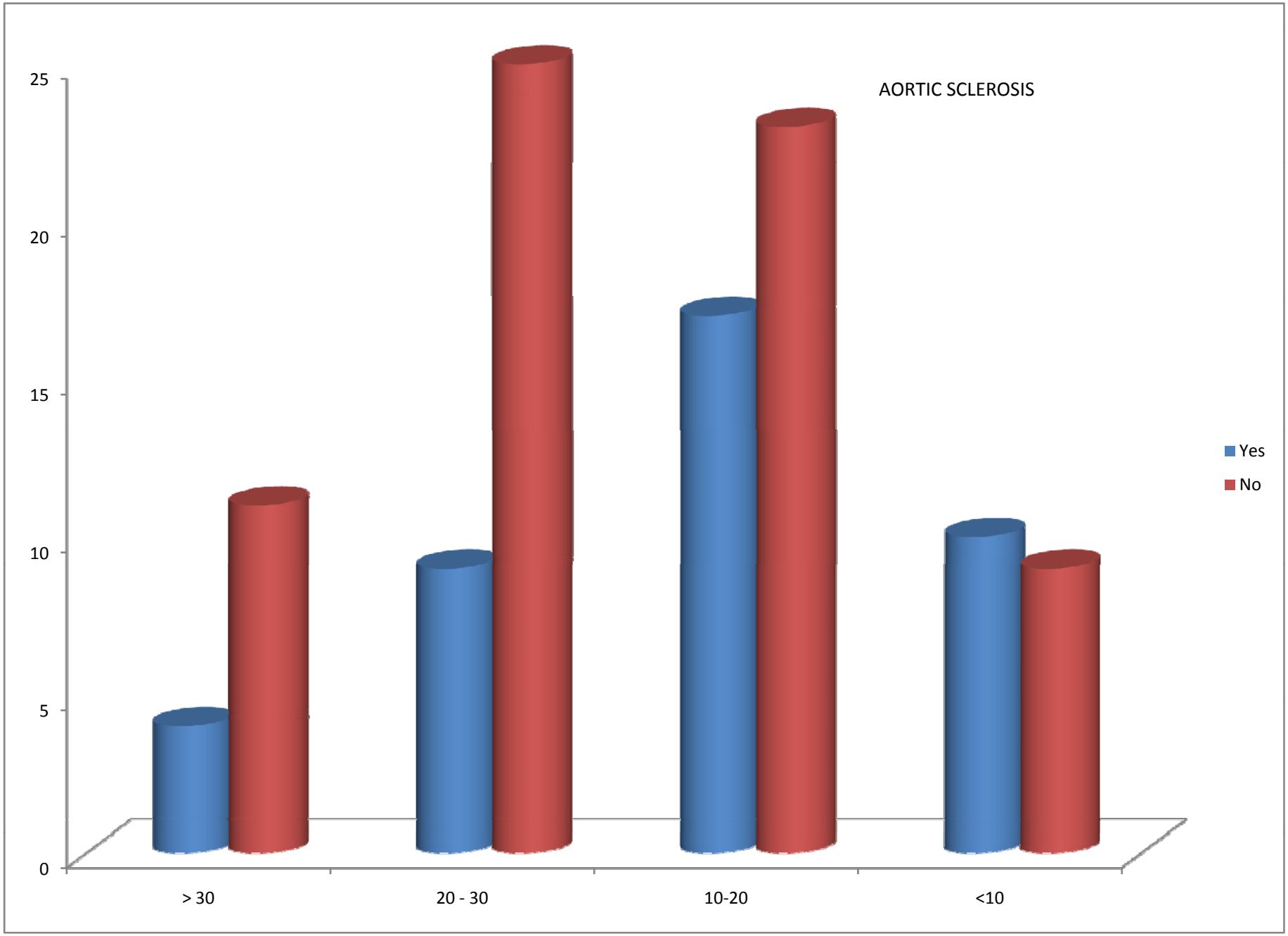
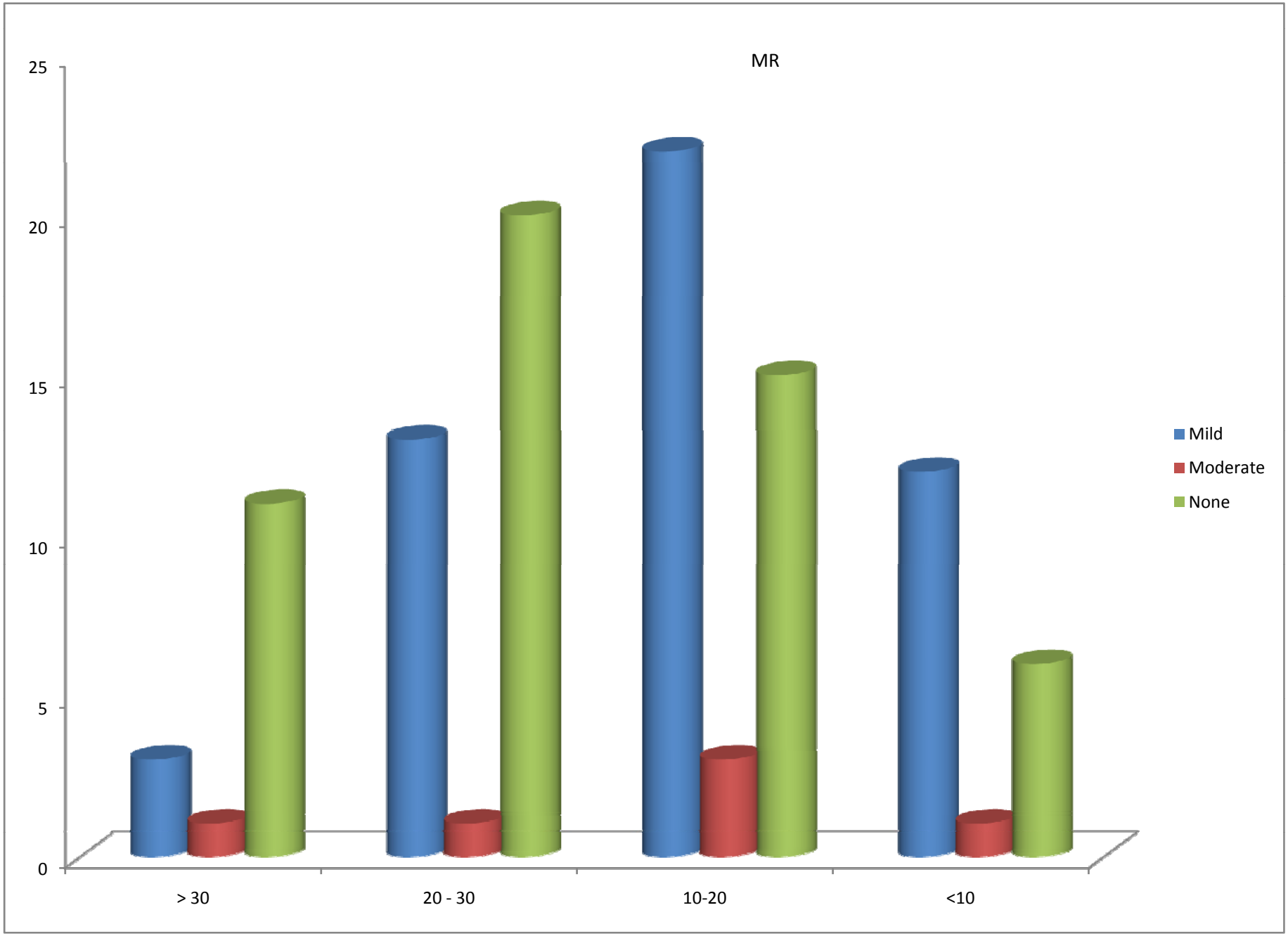
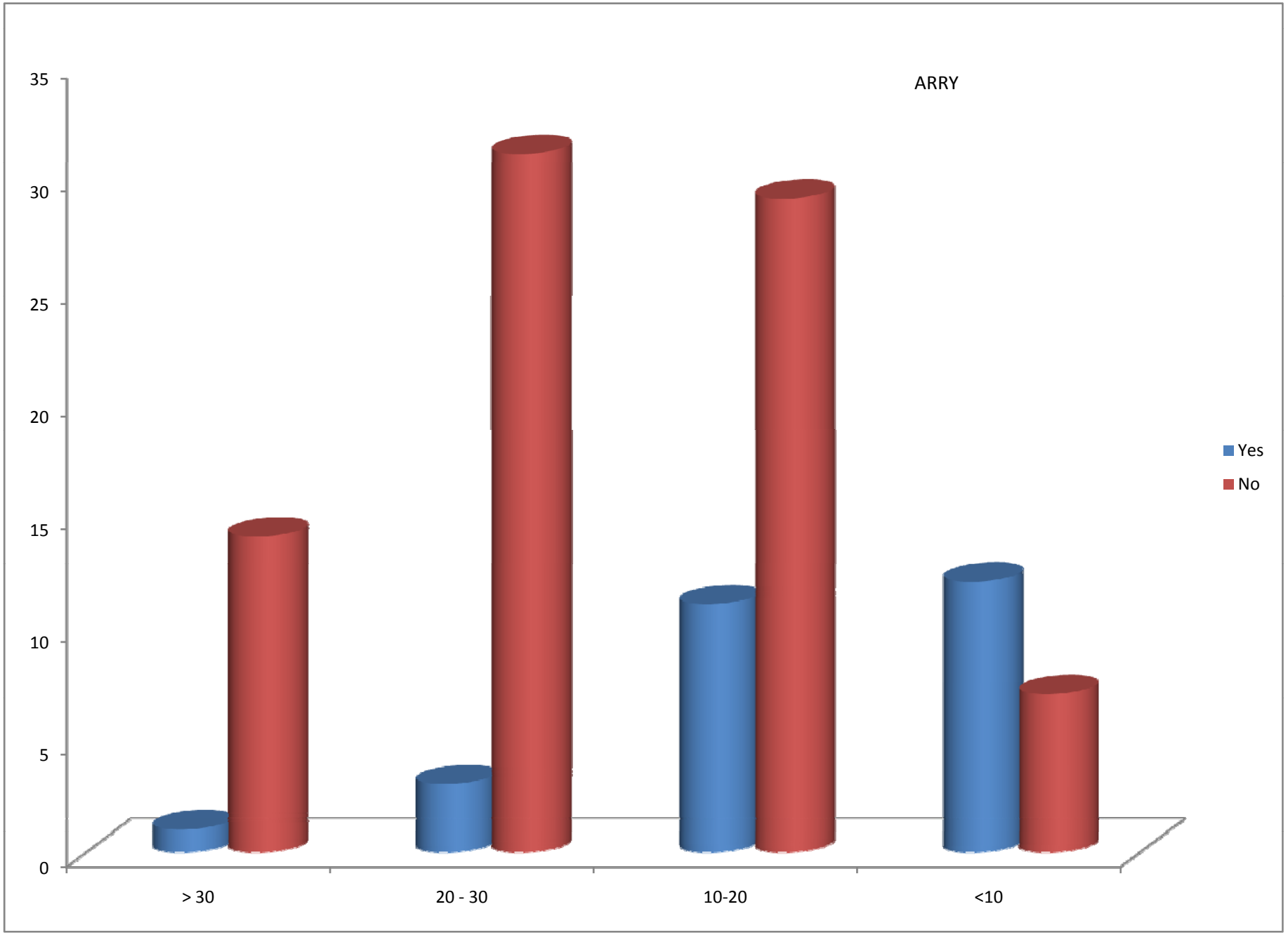
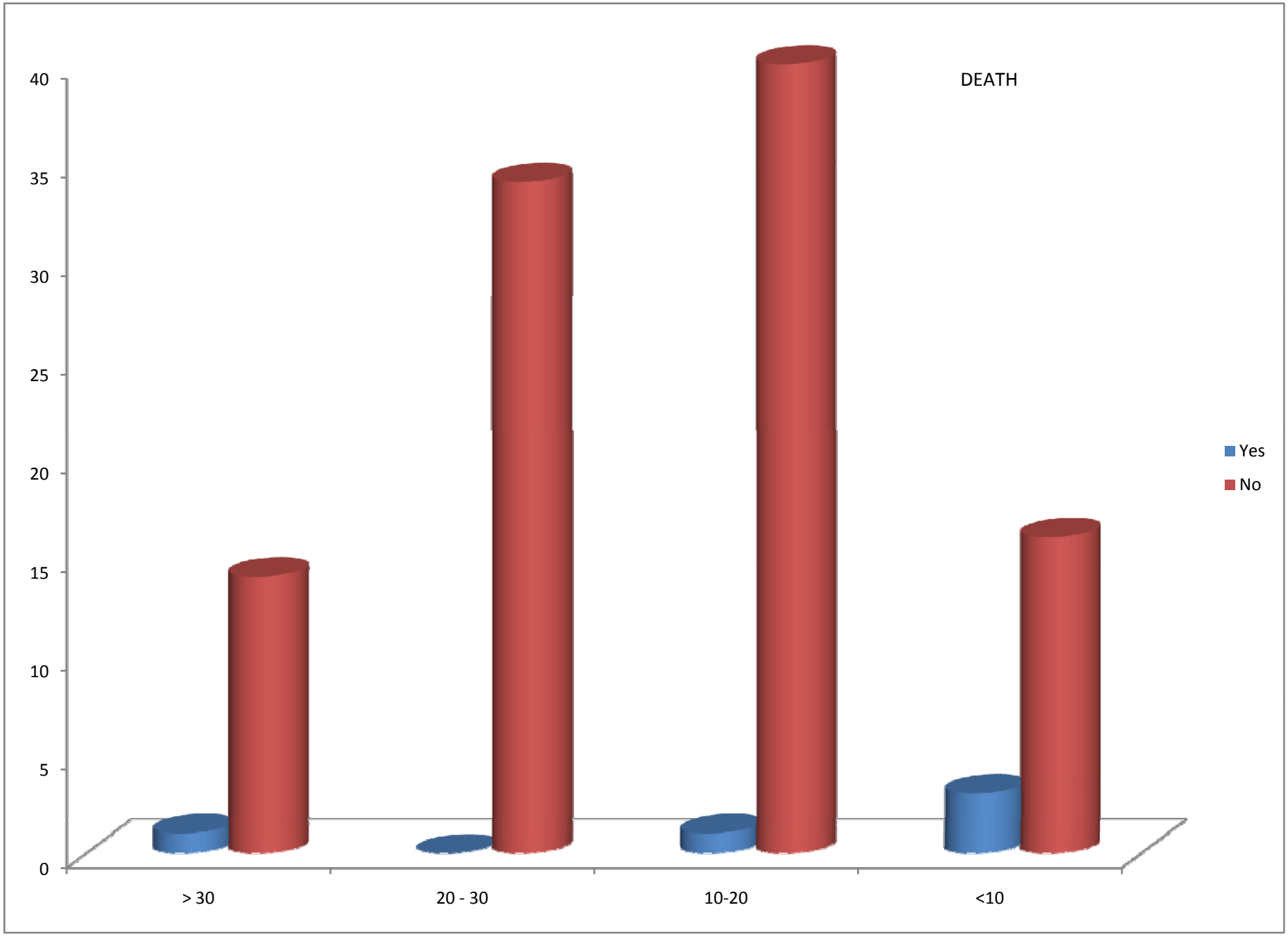

AORTIC SCLEROSIS
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Yes
No

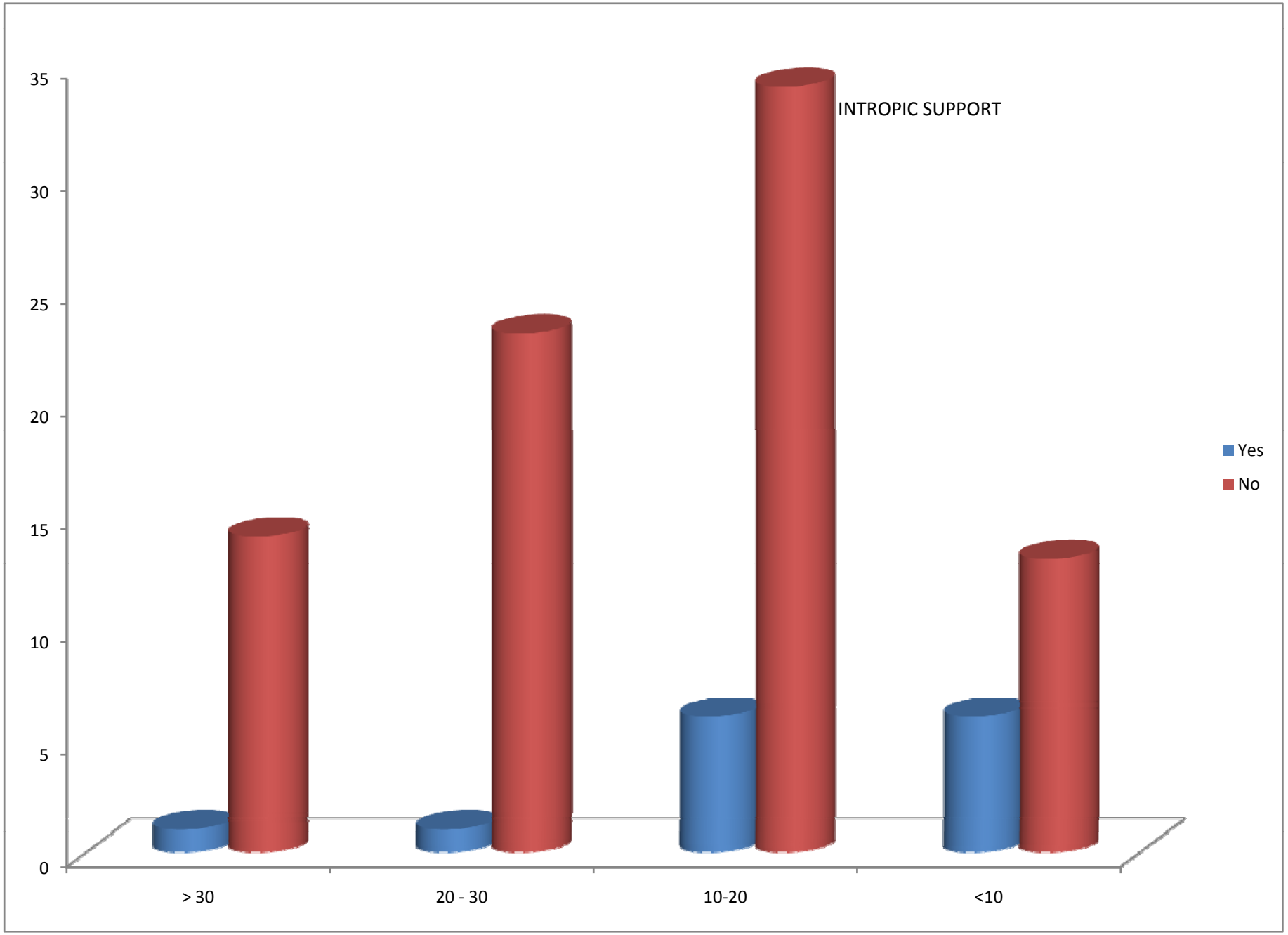

DIASTOLIC DYSFUNCTION
30
25
20
15
10
5
0
Normal
I
II
III
> 30
20 - 30
10-20
<10

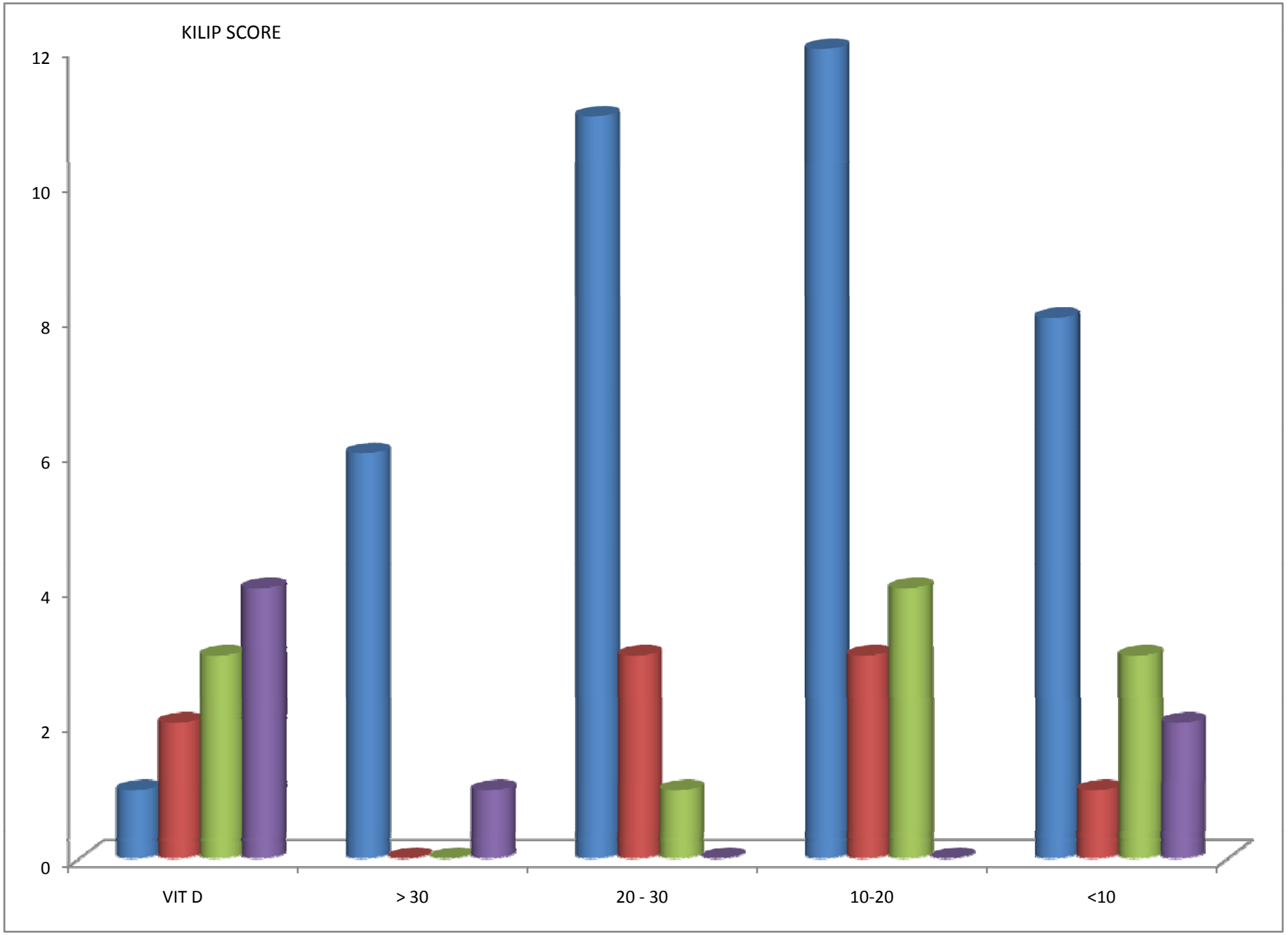

AORTIC SCLEROSIS
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Yes
No

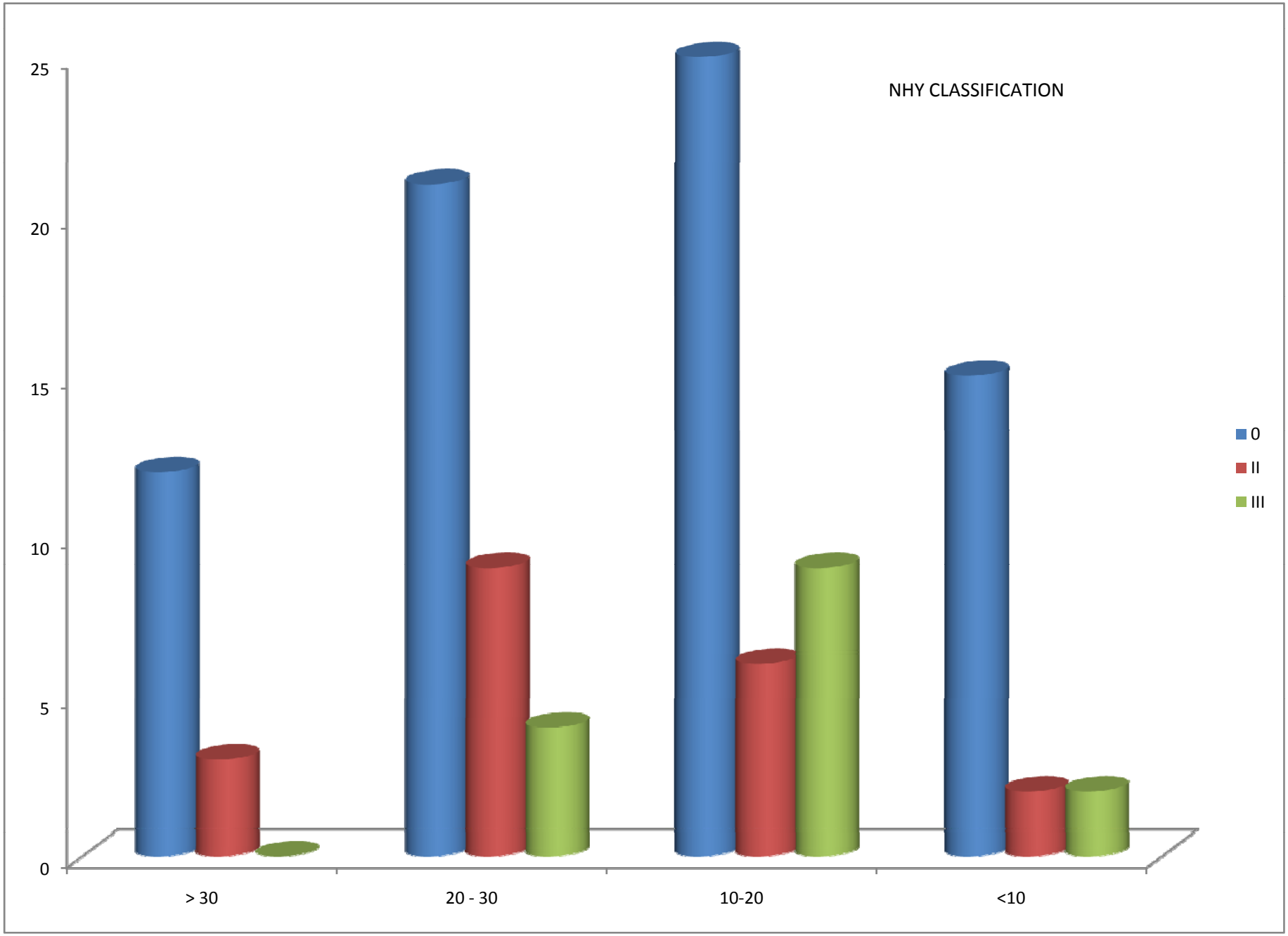

MR
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Mild
Moderate
None

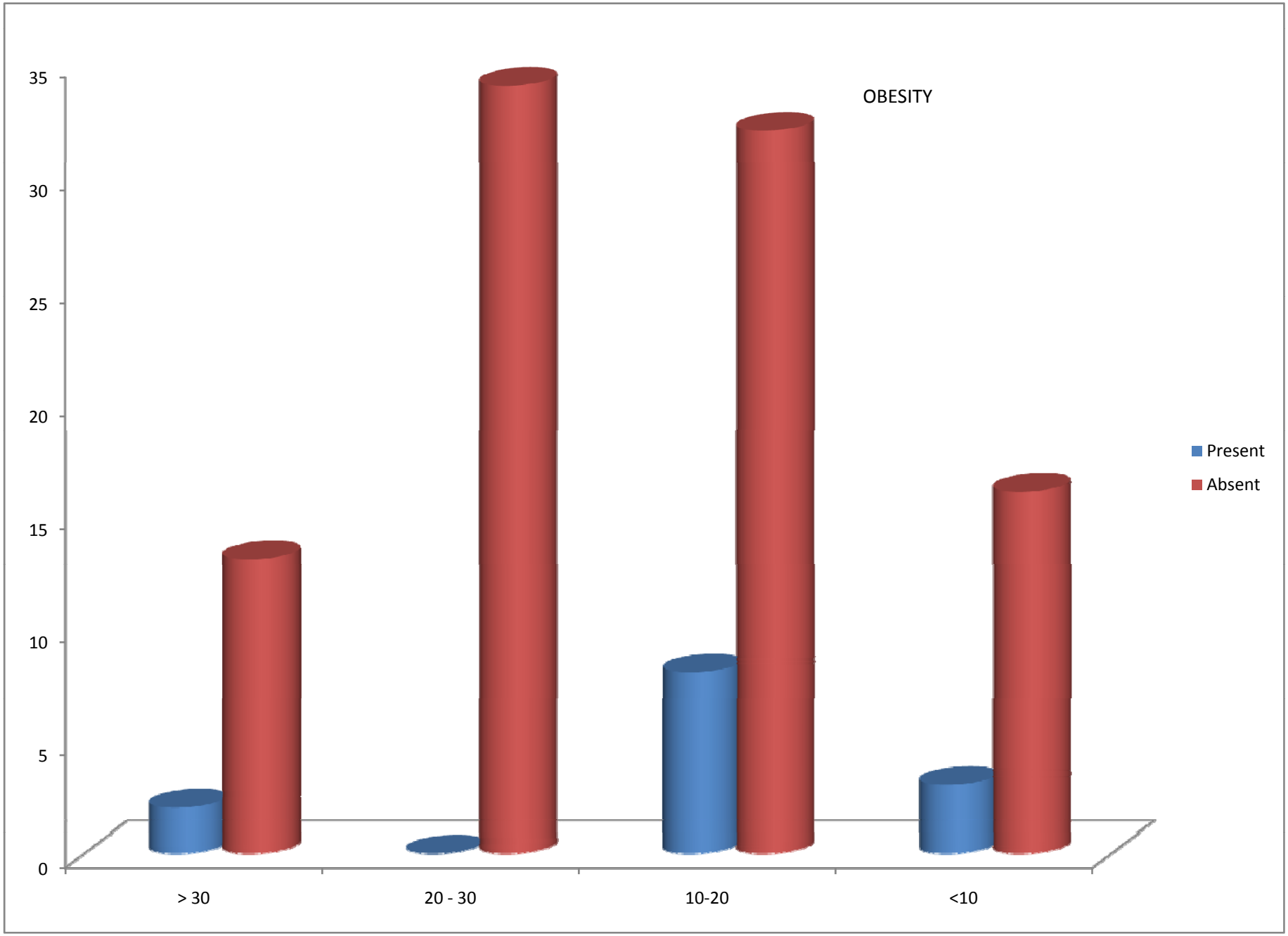

ARRY
35
30
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Yes
No

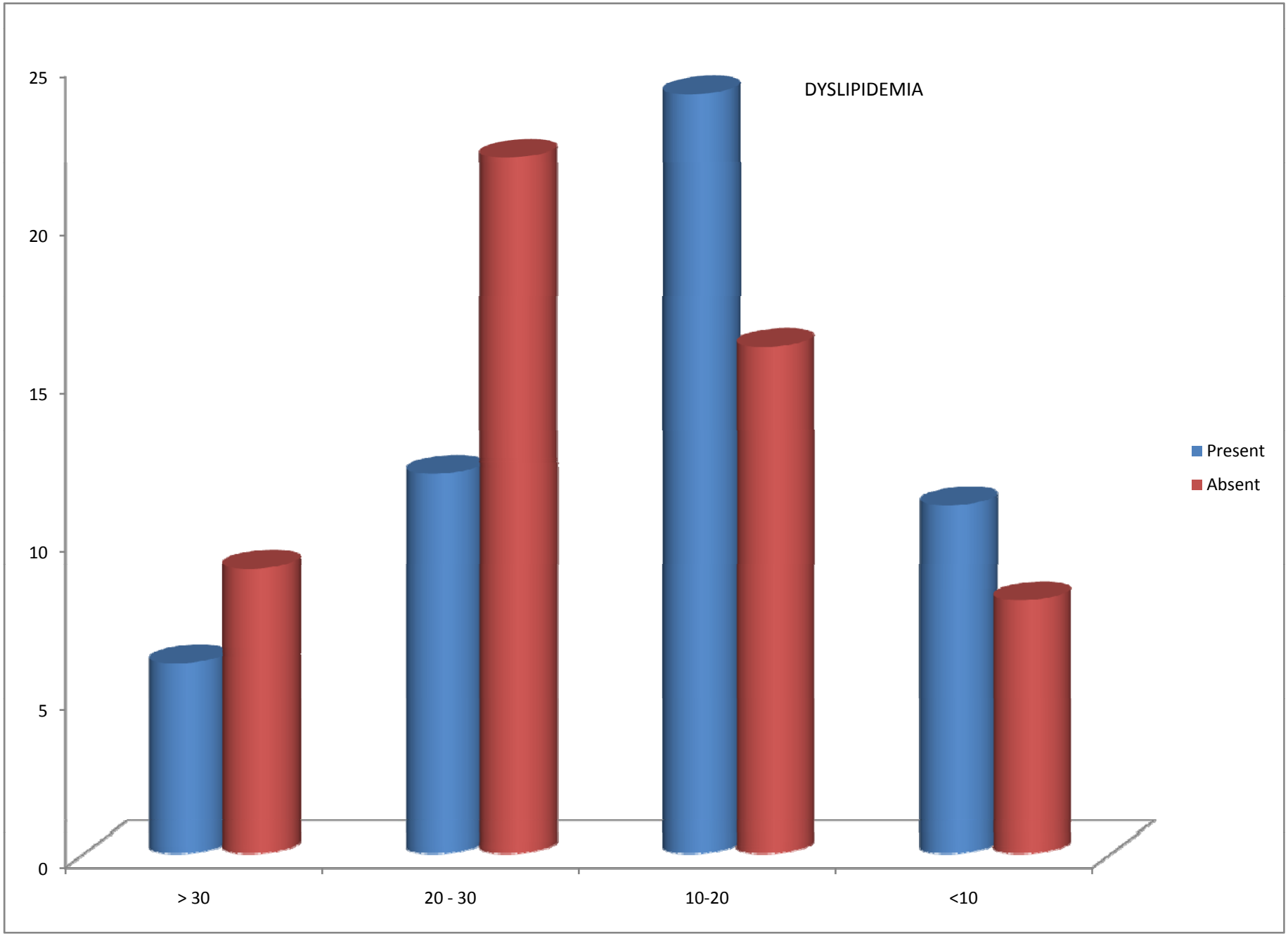

DEATH
40
35
30
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Yes
No

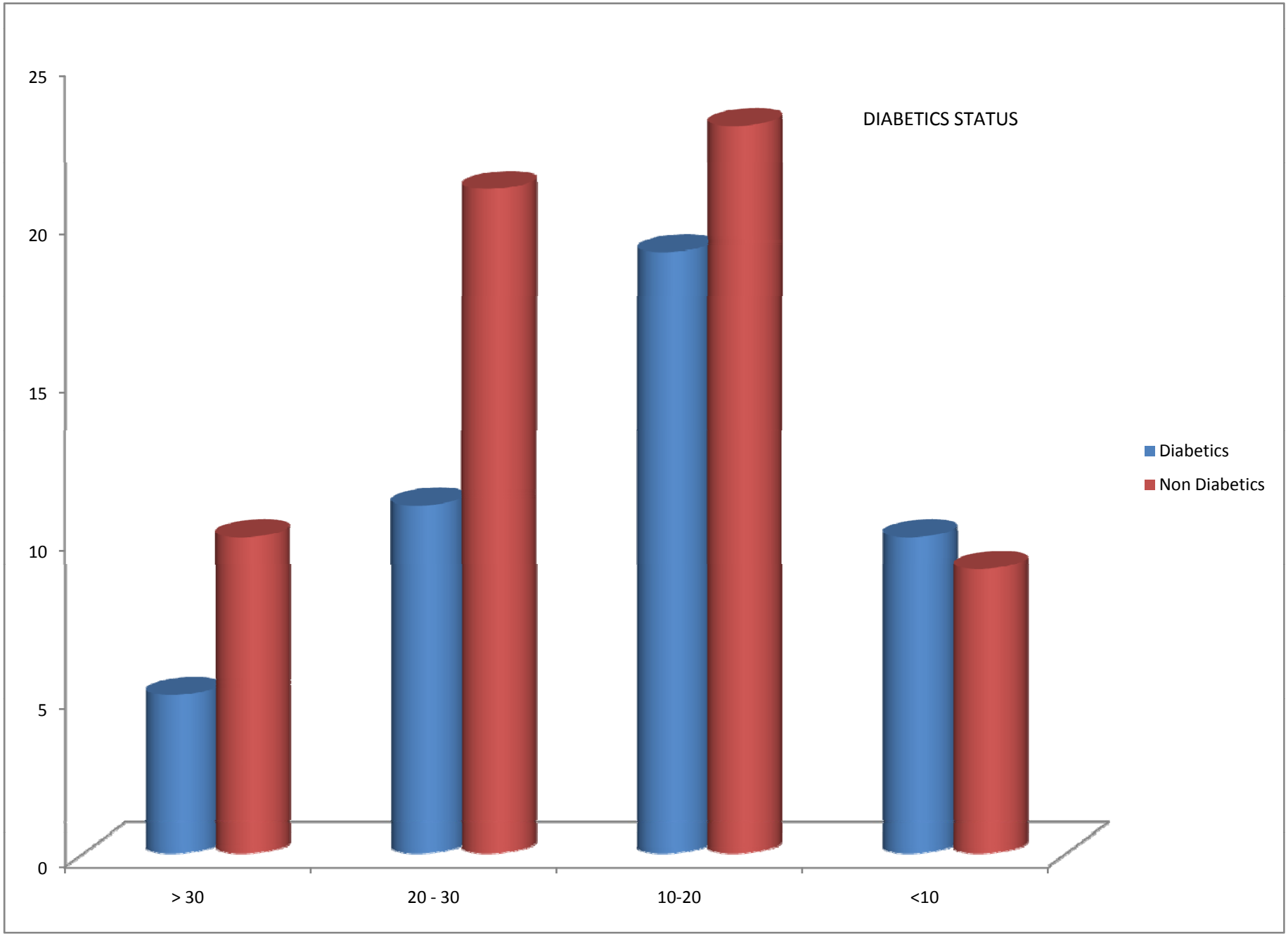

INTROPIC SUPPORT
35
30
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Yes
No

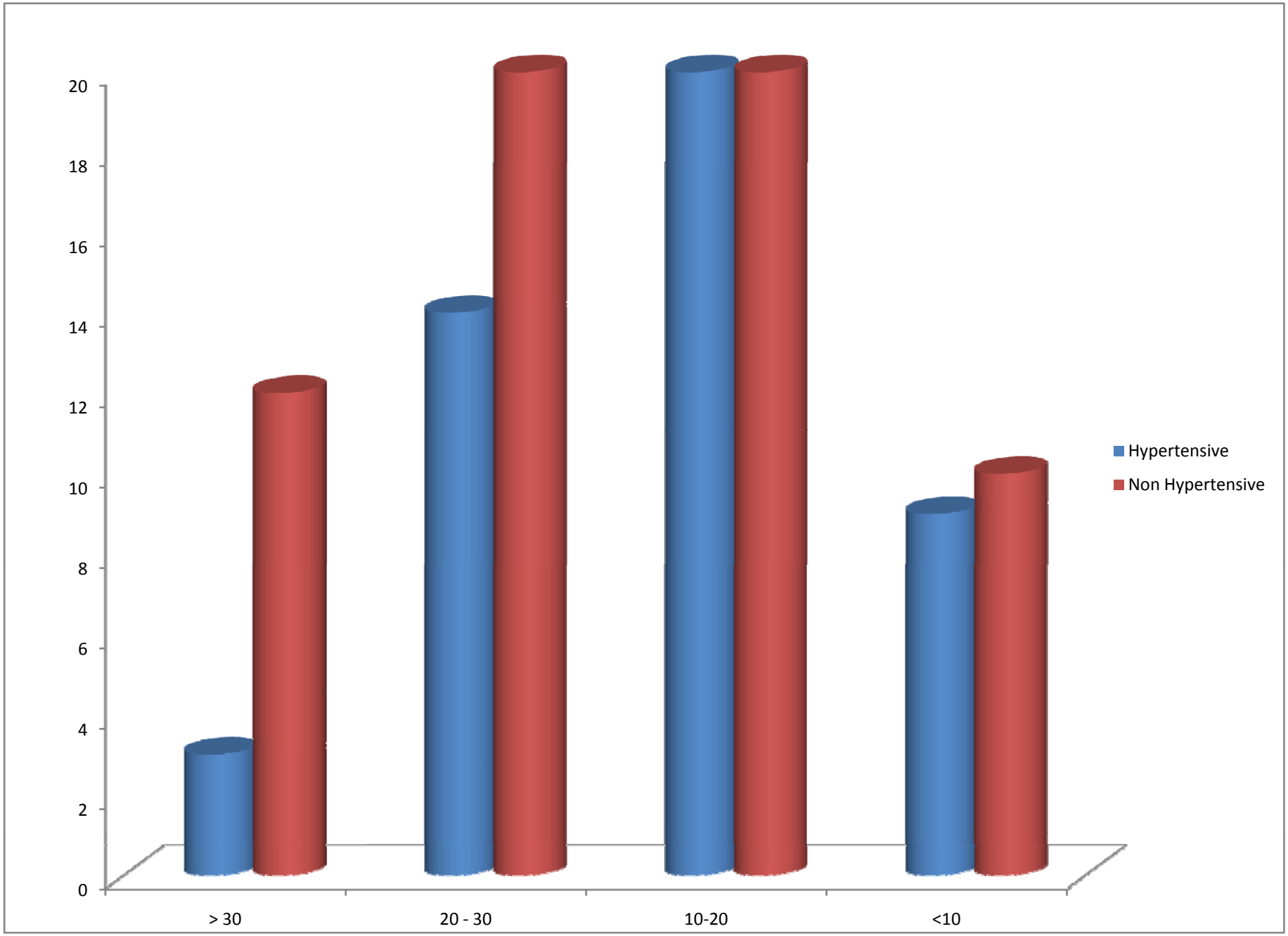

KILIP SCORE
12
10
8
6
4
2
0
VIT D
> 30
20 - 30
10-20
<10

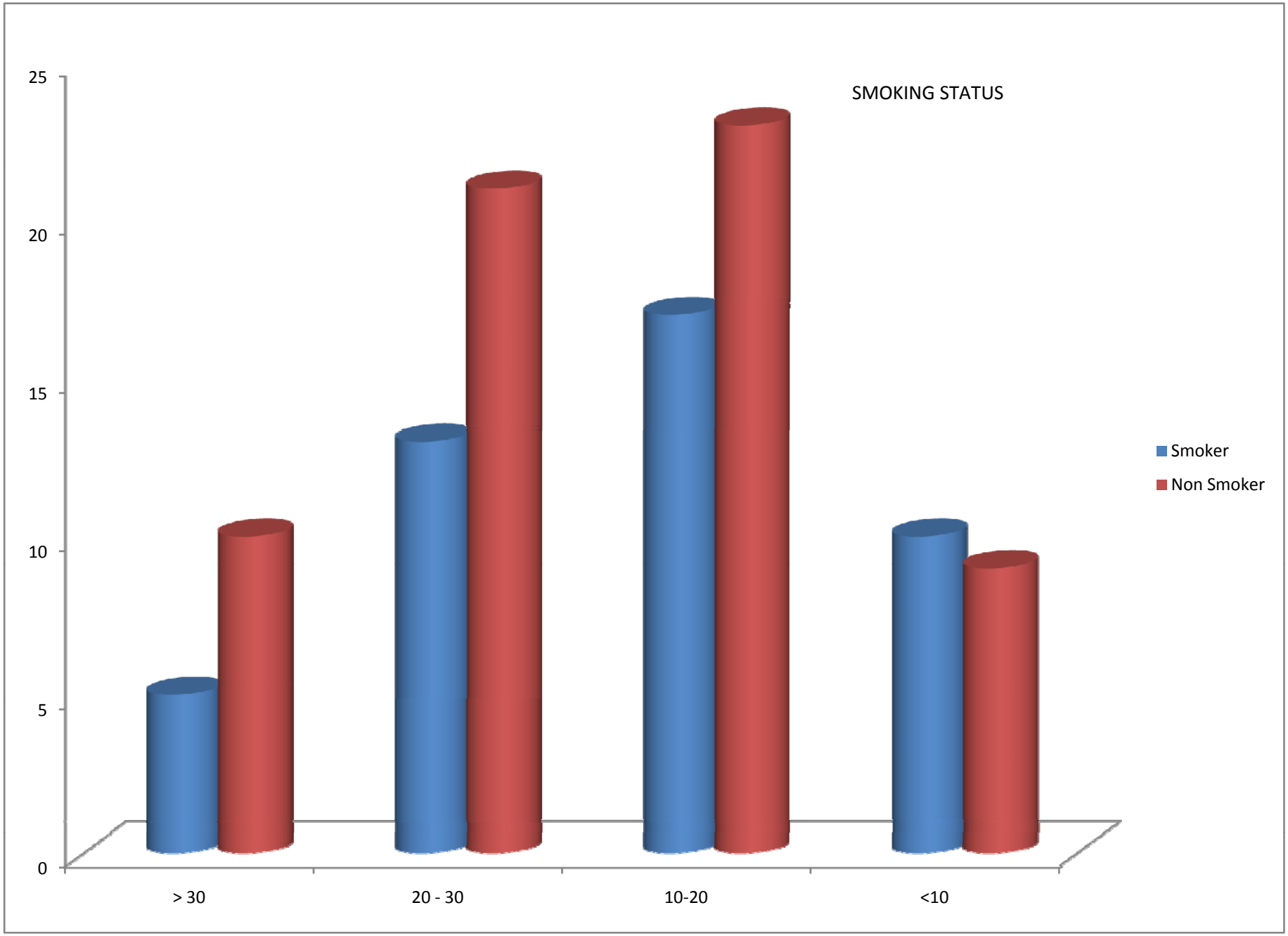

NHY CLASSIFICATION
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
0
II
III

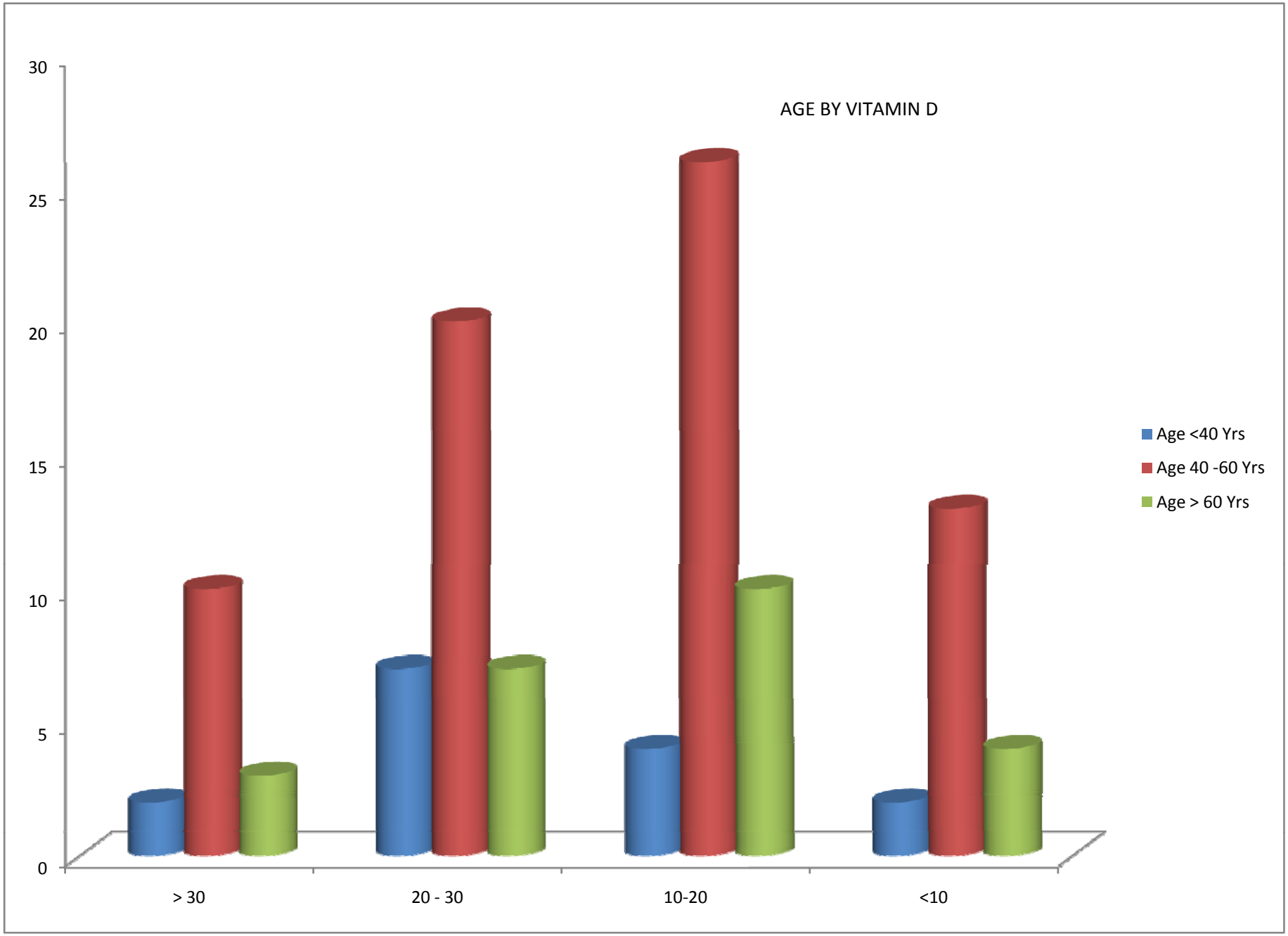

OBESITY
35
30
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Present
Absent

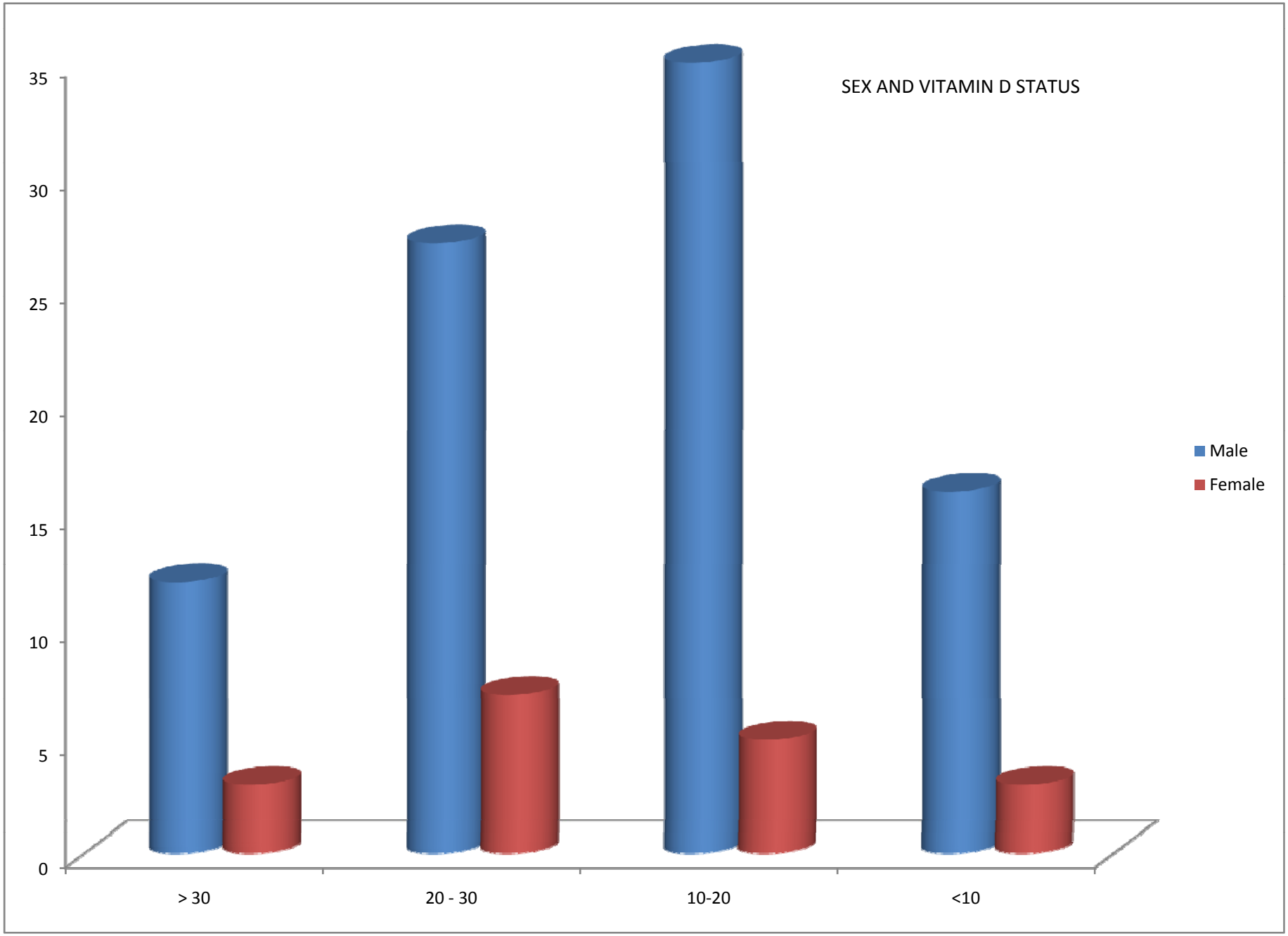

DYSLIPIDEMIA
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Present
Absent

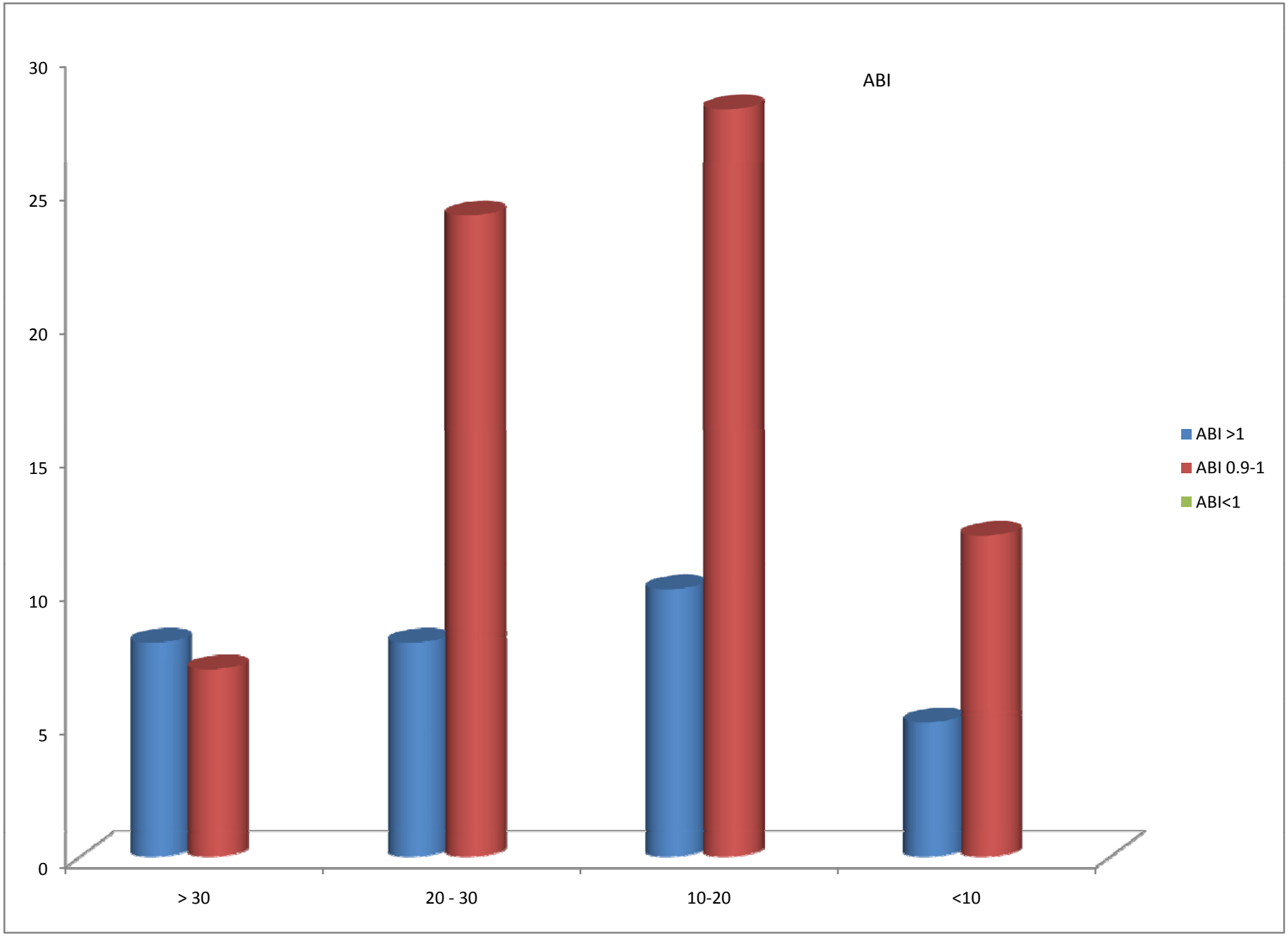

DIABETICS STATUS
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Diabetics
Non Diabetics


20
18
16
14
12
10
8
6
4
2
0
> 30
20 - 30
10-20
<10
Hypertensive
Non Hypertensive


SMOKING STATUS
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Smoker
Non Smoker


AGE BY VITAMIN D
30
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Age <40 Yrs
Age 40 -60 Yrs
Age > 60 Yrs


SEX AND VITAMIN D STATUS
35
30
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
Male
Female


ABI
30
25
20
15
10
5
0
> 30
20 - 30
10-20
<10
ABI >1
ABI 0.9-1
ABI<1